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A TREATISE

ON THE

DISEASES OF THE CHEST:

BEING A

COURSE OF LECTURES

DELIVERED AT

THE NEW YORK HOSPITAL.

BY

JOHN A. SWETT, M.D.

PHYSICIAN TO THE NEW YORK HOSPITAL; MEMBER OF THE NEW YORK PATHOLOGICAL SOCIETY.

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TO

JAMES C. BLISS, M.D.

PRESIDENT OF THE NEW YORK SOCIETY FOR THE RELIEF OF WIDOWS AND ORPHANS OF MEDICAL MEN;

TRUSTEE AND FELLOW OF THE COLLEGE OF PHYSICIANS AND SURGEONS OF THE STATE OF NEW YORK:

In appreciation of his high professional character—and in remembrance of many acts of personal kindness,

THIS VOLUME IS DEDICATED.

PREFACE.

The Lectures on the Diseases of the Chest were published ten years since, in the New York Lancet, a weekly medical journal existing at that time. Being delivered extempore, and taken in short-hand, although by that excellent stenographer, the late Dr. Houston, then Editor of the Journal, they contained necessarily many imperfections, which, under other circumstances, might easily have been avoided. Soon after their publication, however, in this informal manner, the author received repeated solicitations from his medical friends, that he would revise the Lectures and publish them in a separate volume. This request he has complied with in the volume which he now offers to the medical profession. The whole subject has been carefully revised, and the experience of ten years in Hospital and in private practice has been added. During most of his professional life the author has kept a register of the most important cases of chest disease that have fallen under his notice, and these have been numerous and often highly important. He has used them freely in the preparation of the present work, and is happy in believing that his statements are founded upon registered and carefully observed facts, rather than upon vague recollections. His own opinions are embodied in the text: the opinions of authors, as well as such statistical informa-

tion as may serve to illustrate the subject, are contained in notes. Believing in the great importance of microscopic anatomy in the progress of pathology, he has added an Appendix, containing a translation from Lebert's excellent work on Pathological Physiology, of the microscopic discoveries in the structure of tubercle and of cancer, with illustrations from the same work.

New York, Jan. 1852.

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DISEASES OF THE LUNGS.

LECTURE I.

PHYSICAL SIGNS OF PULMONARY DISEASE.

Physical signs of the healthy and the diseased lung.—Signs derived from the inspection of the parietes of the chest: from the touch.—Percussion, the best mode of practising it, and the circumstances which modify it.—Auscultation.—Vesicular murmur, and its modifications.—Rhonchi and rattles.—Vocal resonance; its modifications.

The diagnosis of pulmonary diseases is established by the careful study of three classes of symptoms, or signs. The *constitutional symptoms*, which are the changes produced by these diseases in the general system, and in remote organs; the *rational symptoms*, which are the changes produced by a perversion of the healthy functions, or of the physiological action of the lungs; and, finally, the *physical signs*, which are produced by physical changes in the structure and condition of these organs. It is this latter means of diagnosis to which I wish to call your attention in the present lecture.

The importance of this knowledge is now generally admitted. At the time I commenced the study of these diseases it was in its infancy in this country—those among our Hospital physicians who were not too old to improve, were groping their way in search of light. Mistakes were of course constantly made, and many, even distinguished men, who saw these mistakes, pretended to ridicule an art which they were neither willing to learn nor to appreciate. But this feeling has passed away. Those who do not practise auscultation, now admit its importance, and no physician of the old school would wish his son to be educated in his profession without making himself thoroughly acquainted with the art.

The physical examination of the chest embraces many particulars. Thus, by the *eye alone* you can sometimes gain much valuable information—you can judge, in this way, whether the two sides of the chest, externally, are symmetrical and well developed. If certain portions are *depressed*, or *unnaturally prominent*, this should excite a suspicion of disease. A case of severe pleurisy, with copious liquid effusion into the pleural sac, will illustrate this remark. Thus, when the effusion is copious, the whole affected side is dilated, while after the effusion has been absorbed, the side is contracted. These opposite conditions may also be more partial. In emphysema of the lungs, you will frequently find a limited dilatation of the external walls of the chest, under the clavicle; in tubercular phthisis there is often a depression in the same region. But little benefit would be derived from the study of this physical sign alone. Indeed, it is seldom, even in health, that you will find the two sides of the chest perfectly symmetrical, perfectly natural in their external development. The habits of early education, the occupations of life, by inducing a curvature of the spine, or by directly altering the shape of the ribs, produce depressions and elevations in the parietes of the chest, which are entirely independent of internal disease. Thus, unless these changes correspond with other physical signs, as increased dulness, or increased resonance on percussion, they are of little importance. I may allude here to one important fact. When the parietes of the chest are pushed out by internal pressure, the intercostal spaces are advanced with the ribs, while when the same deformity exists from changes in the spinal column, or in the ribs, the intercostal spaces retain their natural depressions, or these are even increased.

The forms of disease in which we find the walls of the chest dilated, are the early stage of pleurisy, hydrothorax, pneumothorax, in which the dilatation of one side of the chest is general: emphysema, internal tumors, aneurism, or abscess, in which the dilatation is partial.

The forms of disease in which the side is contracted, are the advanced stage of pleurisy, cirrhosis of the lung, in which the contraction is general; tubercles in the lung, atrophy of the lung, in which the contraction is partial.

I may add, also, that when one side of the chest is contracted by disease in the corresponding lung, the opposite healthy lung becomes more developed, and the parietes expanding with this increased development, the contraction is apparently increased, by comparison.

Whenever the parietes of the chest are either expanded or contracted from internal disease, the *movements* of these parts during respiration are usually diminished.

You will also sometimes be able to gain something valuable by the *sense of touch*. The walls of the chest possess a certain degree of elasticity, which they lose when they are in contact internally with any solid or liquid substance, while this elasticity is increased when the quantity of air in the chest is increased.

The elasticity of the parietes of the chest is diminished in pleurisy with liquid effusion, in hydrothorax, in pneumonia, in tuberculous disease. It is increased in pneumothorax, in emphysema of the lungs. When the cause of these opposite conditions is in the pleural sac, these changes are usually noticed all over, at least one side of the chest (pleurisy, hydrothorax, pneumothorax). When the disease is in the lung itself, it is usually partial (pneumonia, emphysema, tubercles).

When the hand is applied to the healthy chest, and the individual is made to speak, a *distinct vibration* is felt. This may be increased or diminished by internal disease. It is diminished by whatever compresses the lung, and especially by effusions into the pleural sac (pleurisy, hydrothorax, pneumothorax). It is increased by whatever condenses the lung without diminishing the calibre of the bronchi (pneumonia, tubercles, dilatation of the bronchi).

But it is the ear which will reveal to you the most valuable physical signs of disease. The *art of auscultation*, as it is called, and which for convenience may be divided into the art of percussion, and the art of auscultation, which latter expression, for the want of a better term, is used to express those physical signs which are revealed by the organs in the chest, by the mediate or immediate application of the ear to the parietes.

Percussion is the oldest discovery, and not at all inferior to its rival, auscultation, in importance. It was at first practised in a

rude manner by Avenbrugger, its inventor, and by Corvisart, who contributed so powerfully to introduce its practice. These distinguished men practised what is called *immediate percussion*. They struck directly upon the walls of the chest with the hand, or in some other way. After a time, however, this method of percussion was entirely abandoned, when Piorry had introduced the method of *mediate percussion*. This method consists in the use of some elastic medium, of moderate size, to which percussion is directly applied. This may be a flat piece of ivory of moderate thickness, or a thick bit of india-rubber, or what is better than either, the fore-finger of the left hand, applied to the chest by its palmar surface. Some practitioners use also a plexímeter, a kind of hammer, but the best plexímeter is the fore and middle fingers of the right hand, with their extremities placed together. Thus you carry always with you the best means of practising percussion.

To understand the art of percussion well, you must have a clear idea of the natural resonance of the chest, of the elasticity of the internal organs, and of the parietes of the chest, on which this resonance depends. It is not the same in all subjects, or in all portions of the chest in the same subject, even in a state of health. The lungs, which are the original source of the resonance of the chest in percussion, are not equally elastic, equally filled with air in different persons. The chest of some individuals resounds, generally, more or less than the chest of other individuals, without it being necessary to account for this by disease. But while this difference may be owing to a natural difference in the development of the lungs, it is much more frequently owing to the condition of the parietes of the chest. If the parietes of the chest are less elastic from the presence of muscle, or from fat, or from ossification of the cartilages, the resonance on percussion will be diminished. If you percuss the chest of a healthy individual, you will find great local differences in the resonance, and these are chiefly dependent upon the accumulation of fat, or of muscle, in particular parts of the chest, or upon the presence of certain solid organs, the heart, the liver, the spleen, which encroach upon the cavities of the chest. A knowledge of this fact has led to the artificial division of the chest into

regions, by certain transverse and vertical lines. But this is not the best arrangement. You have only to remember the medical anatomy of the chest, the situation of the heart, liver, and spleen, the thickness of the muscular substance, and of the fatty deposit in different portions of the chest, to appreciate readily the influence of these causes on percussion. Thus the heart produces a degree of dulness of about two inches square, over the precordial region; the liver, over the lower portion of the right side of the chest, and as far as a vertical line drawn through the nipple on the left side, and extending usually as high as the seventh rib, and sometimes higher. The spleen creates a somewhat indefinite degree of dulness in the lower and lateral portion of the left side, this organ being easily displaced by accidental causes, and varying very much in size, naturally.

It will be evident to you, that whenever the parietes of the chest are covered with fat, this must interfere very much with their resonance. There is but one portion of the chest, the mammary region, in females, which is much exposed to this inconvenience. This region is of no value in percussion.

So, also, when the muscles of the chest are thick, the advantages of percussion are comparatively lost. Thus, you will gain very little from percussion, posteriorly, above the spine of the scapula. There is little or no resonance in this region. So, also, below the spine of the scapula, down to its inferior angle, and anteriorly over the pectoral muscles, the advantages of percussion are quite limited, owing to the thickness of the muscles. Those portions of the chest which resound best on percussion, and therefore give the best indications of pulmonary disease, are the spaces between the clavicle and the third rib in front, the lateral portions of the chest, in and below the axilla, and the posterior portion of the chest, below the inferior angle of the scapula.

While the resonance of the chest, on percussion, is so much dependent on the external muscles, it will be evident to you that the tension of these muscles, by increasing the elasticity of the parietes of the chest, will modify very much the sound on percussion. If the muscles are tense, the resonance is increased; if they are relaxed, the resonance is diminished. Now as one of

the chief advantages of percussion consists in a careful *comparison* of the opposite sides of the chest, and in the same region, as, for instance, in the sub-clavicular regions, if the muscles happen to be tense on one side, and relaxed on the other, this alone will produce a comparative difference in the sound, on percussion. You will recognize, then, the importance of position in the practice of percussion, and of such a position as will allow the muscles on both sides of the chest to be in a state of equal tension.

The best method of percussing a patient is to expose the chest by the removal of all clothing. This is always proper in a male; in females, decency requires that the chest should be covered with some thin material—a common dressing-gown of linen, or of thin cotton, is the most suitable covering. When the chest is exposed, you can judge accurately of its shape and development, and of the different signs furnished by the touch. Let your patient stand up, in an easy and natural position, with his shoulders resting equally against the wall of the room. This is the best attitude for percussing the anterior portion of the chest, especially in cases in which a nice discrimination is required, and it is almost always practicable in such cases. If the patient is more feeble, let him sit in a chair, high enough in the back to support the shoulders. Or if, as may happen, the patient is too feeble to rise from bed, see that all the pillows are removed, and that the patient lies upon the flat of the back, and on a mattress. In percussing the posterior portion of the chest, the patient may stand or sit, with the arms crossed equally upon the breast, with an inclination of the body forward, so as to render tense the muscles in the posterior portion of the chest.

But the position of the patient is not the only thing to be attended to. The practitioner must assume an easy and unconstrained position. He must strike his left fore-finger, which he uses as a pleximeter, a rapid rather than a forcible blow, and use only the movement of the right wrist, keeping the elbow steady. In this way, percussion will be equally performed, without effort, without inconvenience to the patient. Most persons percuss very awkwardly when they commence. They strike too hard: they use the whole arm to give the blow, instead of a smart, yet gentle tap, using the wrist only.

The art of percussion seems to be a very simple thing; you may think that you can practise it perfectly after a little practice. But it is not so. It requires much practice and care to percuss the chest well. Look at those who have practised it for years, and what a difference is there in the results obtained by different practitioners! One will render a slight difference in sound in opposite portions of the chest quite apparent, while another will obtain only the most unsatisfactory results. It is in ascertaining these slighter degrees of dulness, that the tact of the percussor is most valuable.

It is during percussion that you can best estimate the elasticity of the lungs, and of the parietes of the chest. Clearness of sound and the feeling of elasticity go together, and serve to confirm each other, as evidences of a healthy condition.

Sometimes, indeed, when you are doubtful whether any dulness on percussion exists, a want of elasticity will be perceived.

A great advantage in percussion is gained by a careful comparison of the opposite sides of the chest, placed in the same condition as to muscular tension. In cases in which the dulness on percussion is very great and extended, as in pleuritic effusion, or in pneumonia, this comparison is of less importance. But in cases in which the dulness is slight, often almost imperceptible, as, for instance, under the clavicle, in the early stage of the tuberculous deposit, comparative percussion affords most valuable assistance in estimating the degree of dulness.

The natural resonance of the chest on percussion possesses a peculiar tone, which you will readily recognize after a little practice, and the different degrees of dulness are but degrees of the same sound, terminating, in extreme cases, in a perfectly flat sound. Again, when the natural resonance is exalted by a dilated condition of the air-vesicles, the tone is simply increased, but not altered. In certain physical conditions of the chest, however, as where a large and superficial cavity filled with air exists, the tone itself is modified: instead of being pulmonic, it becomes *cavernous*, or like that produced by percussing upon a stomach or intestine distended by gas. In pneumothorax, this is particularly marked. It is also noticed in certain cases in which large and superficial cavities exist in the lungs. It is seldom,

however, that the sound on percussion over the seat of an abscess is as clear as over the healthy lung, while the alteration of tone is often very striking. It is often accompanied by a peculiar *chinking sound*, which the French have compared to the sound produced by striking with the finger a cracked porcelain jar. When the peculiar tone and the chinking sound exist together, the evidence of a cavity can hardly be mistaken. But the chinking sound, heard alone, is not an evidence of a cavity. If the patient is much emaciated, and the intercostal spaces depressed, if the finger you use as a pleximeter is applied across the ribs, instead of in the direction of their course, a small quantity of air may get under your finger, and its sudden displacement by percussion will produce a chinking sound. I have noticed it also in cases in which, under the clavicle, an abundant secretion of mucus existed in the bronchi, without any suspicion of a cavity. Indeed, the tone in these cases is pulmonic, not cavernous.

In practising auscultation, it is not necessary to expose the chest. It should be covered by a soft towel, or by some analogous material, taking care to remove any article of clothing that may create sound as the chest moves, particularly if it be made of silk or of woollen stuff. The existence of fat upon the chest interferes much less with the sounds of auscultation, than with the sound of percussion, and the same fact is still more decided in relation to the muscles of the chest. The thickness of the muscles, and, indeed, their degree of tension, modifies very little the respiratory sounds, so that auscultation may, if necessary, be practised while the patient lies in bed, although I think he can always be most satisfactorily examined while sitting, or while standing.

Immediate auscultation is much better than the use of the stethoscope. Expose the chest of your patient, or cover it only by a soft towel, and apply your ear directly to the parietes. You will hear the respiratory sounds more distinctly and more satisfactorily than with any form of the stethoscope. In certain cases, in which from malformation of the chest, or from an inability of the patient to assume a convenient position, or when you wish to circumscribe a local lesion, as, for instance, an abscess in the lung, the stethoscope may aid you. A flexible stethoscope is perhaps the most convenient form of the instrument.

I wish to call your attention to a few facts in what may be called the medical anatomy of the lungs, before I attempt to explain to you the physical signs furnished by auscultation. The great mass of the lungs is, as you know, composed of minute air-cells, which are united into lobules, each of which may be regarded as a lung in miniature. Each lobule has its own independent bronchus; all its air-vesicles communicate with each other, or with this bronchus, and it is entirely separated from the surrounding lobules by cellular tissue. The bronchi in their first divisions, more or less rigid and cylindrical from the presence of cartilaginous rings, gradually lose this peculiarity, and become easily compressible tubes, composed of a fibrous and muscular coat, and lined by a delicate muco-serous membrane, which is continued into the air-cells. These bronchi, together with the pulmonary vessels, ramify in the cellular tissue between the lobules; but each terminal bronchus, and there is one for each lobule, plunges into the centre of its lobule, and terminates there in a slight enlargement. Around this terminal bronchus the air-cells of the lobule cluster. Those in contact with it open directly into it; those more remote, open into other air-cells nearer the bronchus. Thus the air which passes through the bronchus on inspiration, does not distend all the air-cells of the lobule at the same moment; for the air must pass through a succession of cells communicating with each other, before all are filled by it.

If you apply your ear to the healthy chest, you hear, during inspiration, a soft, prolonged, swelling murmur, ceasing with the dilatation of the chest. At the commencement of expiration, the same murmur returns, but less distinctly. It is also of much shorter duration, ceasing when about one-third of the act of expiration is completed. These two murmurs, the prolonged and distinct murmur of inspiration, and the more feeble and short murmur of expiration, constitute *the healthy, the natural respiratory sounds.* These murmurs are produced by the friction of the air against the inner surface of the air-cells, as it passes in and out of these cells during respiration.

The distinctness of this respiratory murmur is not the same in all persons. There are natural differences which are permanent, and extend to the whole of the lungs. In some persons,

the respiratory sounds are unusually distinct; in others, again, they are more feeble. Accidental causes, such as agitation of mind, will sometimes render the respiratory murmur very indistinct, for the moment, all over the lungs; and if you request such patients to expand the chest forcibly, this only adds to the difficulty. When all agitation and effort have ceased, then the respiratory murmur becomes more distinct.

When the respiratory murmur is unequal in different portions of the lungs, feeble in one part, unusually distinct in another, this may be regarded as an indication of disease. Feebleness of the respiratory murmur arises from two opposite conditions of the lungs—compression of the air-cells, as in pleurisy, or in the deposit of miliary tubercles; or from a dilated and rigid condition of the air-cells, as in emphysema. In the former case, the air cannot readily enter the air-cells; in the latter, it cannot readily escape from them: so that, in either case, the friction of the air against the cell-walls is diminished, and the respiratory murmur of course is feeble, or in certain cases is entirely lost.

In children, the respiratory murmur is naturally very distinct: hence, in adult life, an unusually distinct murmur is called *puerile*. In old age, the respiratory murmur gradually grows more feeble. In childhood, the lungs are very active; the air-cells, numerous and in a healthy condition, are readily filled with air. In old age, the air-cells become less numerous; they break down into larger, irregular cells: thus the respiratory murmur becomes feeble from atrophy of the lungs.

A puerile respiratory murmur, if confined to a limited portion of the lungs, becomes an indirect indication of disease. It indicates imperfect respiration in some other portion of the lungs.

Sometimes, the murmur of inspiration, instead of swelling on the ear in a continuous sound, is *interrupted or jerking*. This condition is owing to the existence of some cause which prevents the equal and steady dilatation of the air-vesicles. This cause is, perhaps always, the limited deposit of miliary tubercles in the lungs. These are the changes which the true respiratory murmur, produced in the air-cells, undergoes.

Let me now call your attention to the *bronchial sounds*, to

those generated by the passage of the air along the inner surface of the bronchi.

In a perfectly healthy condition of the lungs, the sound generated by bronchial friction does not reach the ear, it is lost in the more superficial murmur of the air-cells. But let any cause exist, which shall either alter the natural proportion between the number and size of the bronchi and of the air-cells, or exaggerate the bronchial friction, or diminish the murmur in the cells, then the bronchial sound begins to be heard. The bronchial sound has three characteristics, as distinguished from the vesicular murmur. The sound of expiration tends to become as long and as distinct as that of inspiration: both sounds are harsh, and when well developed, blowing in their character. Thus when the bronchi are large and numerous, in comparison with the air-cells, this prolongation of the expiratory sound occurs. *It exists naturally at the summit of the right lung, where this condition of the bronchi exists.* It is produced accidentally by a dilatation of the bronchi, by an obstruction of the air-vesicles in a portion of the lungs, as by a slight tuberculous deposit, or by a lobular pneumonia. The sounds of respiration are harsh when they reach the ear from the bronchi. The simplest illustration of this fact is found in the first stage of bronchitis, when the secretion of mucus has ceased, and the bronchial tubes are dry and rough. Then, the friction in these tubes by the passage of the air becomes increased, and the sound, rendered more distinct, reaches the ear in spite of the vesicular murmur, with which it is blended, and to which it gives a certain harshness of sound. When, again, the air-vesicles are partially obstructed, or permanently dilated, and cannot generate much sound, then the harsh bronchial respiration is also heard, mixed with the feeble vesicular murmur, as when the lung is affected by miliary tubercles, or by emphysema. Finally, if no air enter into the vesicles of a certain portion of the lung, and the pulmonary tissue around the bronchi is condensed and solidified, so as to readily communicate the vibrations of the bronchi to the ear, and without any mixture of vesicular murmur, then the completely formed bronchial respiration is heard, with its distinct and long expiratory sound, its harshness, its blowing char-

acter, as in the second stage of pneumonia, in tuberculous infiltration. The same sound also occurs in dilatation of the bronchi with compression of the surrounding pulmonary tissues.

If a cavity form in the lung, communicating freely with a bronchus, and this cavity becomes emptied of its fluid contents, the air passes freely in and out during respiration, and the vibrations generated by the friction of the air against the walls of the cavity, resounding in a hollow space, produce a hollow sound during both inspiration and expiration, less harsh usually, and often less distinct, than the bronchial respiration. *This is the cavernous respiration*—which, when the cavity is very large, and with thin and elastic walls, sometimes assumes a clear, ringing tone, which modification of sound is the *amphoric respiration*—the sound being like that produced by blowing into an empty flask. The cavernous respiration exists, sometimes, in dilatation of the bronchi, but most frequently in tuberculous abscesses, and in pneumothorax.

These modifications of the respiratory sounds, although very distinct from each other in their well-marked forms, the vesicular, the bronchial, and the cavernous respiration, yet pass into each other by insensible gradations, so that sometimes it is difficult to distinguish the one from the other. The changes in the vesicular murmur, it being loud, or feeble, or jerking, are often mixed with the first degrees of the bronchial respiration, the prolonged expiration, the harsh sound; thus producing a modified respiratory sound, which may still retain something of the vesicular character. Thus it may be feeble, with a prolonged expiration, or with harshness, until gradually, with the changes in the condition of the air-cells, it gradually assumes the character of the pure bronchial respiration, just in the same manner as the bronchial respiration passes into the cavernous respiration by almost insensible gradations. I should remark, that at the root of the lungs, especially in thin persons, there is naturally a bronchial respiration over a limited space — at the point where the large bronchi enter the lung, and that a naturally cavernous respiration exists over the trachea and larynx. Thus, by practising auscultation upon each other, you can easily learn to distinguish the different modifications of the respiratory

sounds as produced by disease. This practice I would especially recommend to you.

The bronchial tubes, as well as the air-cells, are lined by a delicate membrane, mucous in the larger tubes, but gradually becoming more serous in its nature as it approaches the air-cells. This membrane secretes in health a thin mucous or muco-serous fluid, which is sufficient to lubricate its inner surface, and to preserve its smoothness and softness. When this membrane is inflamed, the first effect of inflammation is to stop this secretion, and the membrane becomes dry. The effect of this is to render the respiratory sound harsh, and to prolong the expiratory sound by developing, in a slight degree, the bronchial sound; for the bronchi have lost something of their natural smoothness, and consequently the air which passes over their internal surface produces stronger vibrations. But this is not the only effect produced: the bronchi are rendered irritable by the existing inflammation, and being muscular tubes, at least in the smaller branches, they contract spasmodically at certain points, and the air passing through these narrowed portions produces a sort of whistle or chirp, which is called the *sibilant rhonchus*. This sound is heard in the smaller bronchi. In the larger tubes, in which the spasmodic contraction is less, owing perhaps to the presence of the cartilaginous rings, and to the less perfectly developed muscular coat, as well as to the larger size of the tubes, the sound becomes much more grave and sonorous, a cooing sound, or like the notes of a bass-viol. This is the *sonorous rhonchus*. It has been supposed, also, that a small portion of viscid mucus, adhering to the inner surface of a bronchus, and partially obliterating its cavity, may generate these sounds; but this is a doubtful, and at all events, not a frequent cause of their existence.

These rhonchi, like other spasmodic effects, are transient and intermittent in their character. Whatever overcomes the muscular contraction, the spasm, causes them to cease. Thus the act of coughing will do this temporarily; but it is only when the irritability of the tubes is diminished that these sounds cease permanently. It is in the first stage of bronchitis that these sounds are heard, and also, more permanently, in cases of

emphysema, a disease which is usually accompanied by an irritable condition of the bronchi. It is sometimes, also, noticed in tuberculous disease of the lungs.

An increase of the secretion of the lining membrane of the bronchi and of the air-cells is also of very frequent occurrence. In the air-cells, it is a thin serous fluid; in the bronchi, it is a more viscid secretion. This secretion, when acted upon by the air, which comes into more or less violent contact with it during its passage through the lungs, forms air-bubbles of various sizes, and of different degrees of resistance. In the air-cells, these bubbles are necessarily minute: they are equal in size, and only capable of being formed and ruptured during the more forcible action of the air in inspiration. These bubbles, also, are incapable of being displaced, being inclosed in a small cavity. Thus you have a rattle formed by the bursting of these bubbles —fine, equal, heard only during inspiration, bursting from a circumscribed spot under the ear by a sort of explosion, especially at the end of a full inspiration. This is the *crepitant rattle*, the rattle heard in the first stage of pneumonia, and in certain cases of pulmonary œdema, when serum is effused into the air-cells.

The secretion into the bronchi produces a different rattle. The secretion is viscid, but unequally so. The current of air has a more or less free passage backward and forward through these tubes; consequently, the bubbles produced by the action of the air are larger, but unequal in size: they are formed, and they break both during inspiration and expiration, and they appear to travel along the tubes, forward and backward. You have then a rattle, coarse, unequal, heard during inspiration and expiration, movable. This is the *mucous rattle.*

In the smaller bronchi, in those near their terminus, the secretion approaches in character that of the air-cells, and the tubes are more minute. This produces a rattle approaching in fineness and in equality of sound the crepitant rattle, but still heard during inspiration and expiration. This is sometimes called the *subcrepitant rattle.* Again, when the secretion is very abundant, and in the larger tubes, as in the trachea, in a dilated bronchus, or in an abscess communicating freely with a

bronchus, this rattle becomes so loud and so abundant as to produce a gurgling sound.

In certain cases, in which a very large and superficial cavity exists, containing a fluid, and communicating with a bronchus, the mucous rattle assumes a metallic sound, which is commonly called the *metallic tinkling*. This metallic rattle is produced in different ways. Sometimes, it is produced by the bursting of bubbles of air on the surface of the fluid contained in a cavity partially filled with air; at other times, it seems to be produced by the transmission of the sound, produced by the breaking of air-bubbles in the lungs, through a large cavity containing air, but not communicating with the lungs; and finally, it is caused by the dropping of fluid from the walls of a large cavity into a mass of fluid, the surface of which is in contact with a body of air.

Let me illustrate this rather complicated statement. Suppose a case of what is called hydro-pneumo-thorax, in which a communication exists between a bronchus and the pleural sac. In this case, the lung is compressed, and the pleural sac is converted into a large cavity containing air and fluid, the walls of which are very elastic, which is an essential condition to the production of the metallic sound. The air, in passing through the bronchus which opens into this cavity, passes through the liquid in the cavity, and in its passage forms bubbles which break in the air in the cavity above this fluid, with a metallic sound. This is the explanation of this sound in perhaps a majority of cases. Again, the pleural sac is distended only with air—it is simple pneumothorax; no communication exists between this great air-chamber and the bronchi. But a mucous rattle exists in these bronchi which reaches the ear, the vibrations being transmitted across the great air-chamber formed by the pleural sac. This also will give to the rattle a metallic character. Finally, air and fluid both existing in the pleural sac, and the patient being in a recumbent position, the fluid, by gravity, will mount to the upper wall of the cavity corresponding to the apex of the lung. When the patient suddenly sits up, the fluid subsides again to its lowest level, and the air fills the space above it; but a few drops of the liquid adhere, for a moment, to the

superior wall of the cavity, and then fall off into the fluid below. Each drop as it strikes the fluid produces a metallic sound, which is reverberated by the walls of the cavity.

A metallic splash, if I may use the term, is also produced by shaking the patient sharply by the shoulders when the pleural sac is filled with air and fluid. This is commonly called the Hippocratic succussion, for it was known to the father of medicine.

These different rattles, like the modifications in the respiratory murmur, all pass into each other by insensible gradations. Distinctly marked in most cases, it is sometimes difficult to determine the precise character or shade of sound; neither is this very important.

These rattles can also be imitated artificially. The crepitant rattle may be imitated by throwing fine salt in the fire, or by rubbing the hair over the temples between the fingers; the mucous rattle by blowing soap-bubbles; the metallic tinkling even may be imitated, by taking a bladder distended by fluid and air, and passing in air forcibly by a syringe, which shall communicate with the cavity, below the level of the fluid.

If you apply the ear to the chest, and request the patient to speak—the best method is to request him to count one, two, three, slowly and distinctly, in his natural tone of voice—vibrations of sound are transmitted, from the larynx to the bronchi, and through the parietes of the chest, which strike the ear with a certain shock. This is the *vocal resonance*. There is naturally more resonance at the summit of the right lung than at the summit of the left lung, for the reason that the bronchi are larger, shorter, and more direct in their course in this portion of the lung. If the bronchi are dilated, if the pulmonary tissue around these tubes is condensed, solidified, as by pneumonia or by the deposit of tubercles, or if a tumor, as an enlarged bronchial gland, presses upon a bronchus on one side, and on the parietes of the chest on the other side, the vocal resonance is increased, and constitutes what is called *bronchophony*. In certain cases, in which abscesses exist in the lung, which are superficial, filled with air, and communicating freely with a bronchus, this resonance is still more distinct. It sometimes strikes the

ear with a shock that is almost painful, and the voice seems to come directly from the cavity. This is called *pectoriloquy*. In cases, in which a very large and superficial cavity exists, also communicating freely with a bronchus, as in pneumothorax with perforation of the lung, the vocal resonance has a metallic ring, which is called the *amphoric resonance*. These variations in the vocal resonance are but degrees of the same sound, and pass, by insensible gradations, into each other.

I am not disposed to attach a very great degree of importance to this vocal resonance. Because, in the first place, there is no standard of the natural resonance of the voice. It varies very much with the tone of the voice in different individuals, being more distinct in those whose tone of voice is bass, less distinct in those who speak in a higher key. It is consequently more distinct in men than in women. Indeed, the same individual may alter it at pleasure by speaking in a higher or lower key. The important fact, however, that the vocal resonance is naturally more distinct at the summit of the right lung than at the summit of the left lung, must be carefully remembered, especially when you wish to judge of slight differences in the vocal resonance in these situations. The vocal resonance is diminished by whatever compresses the lung, and especially by large effusions of liquid into the cavity of the pleura.

There is, however, one form of vocal resonance which is much more important, because it is not a difference in degree, but a difference in kind. It is what is called *egophony*, from its resemblance to the bleating of a goat, or, what we understand better in this country, the bleating of a sheep. It is a clear, silvery sound, in a higher key than the voice, and strikes the ear in an interrupted manner, and with very little impulse. It is heard when there is a moderate effusion of liquid in the pleural sac, as in pleurisy and in hydrothorax. It seems to be produced by the vibrations from the bronchi being transmitted to the ear over the surface of the liquid effusion, like the ripple on the surface of a pool of water from a puff of wind. It is usually noticed most distinctly along a line drawn from the lower angle of the scapula around the affected side. If the lung is much compressed by a large effusion, this sound is not heard.

In cases of liquid effusion into the pleural sac, if the lung is held partially to the walls of the chest by old pleuritic adhesions, so that its surface only is compressed, or in cases in which the compression of the lung is only partial from a solid deposit in its substance, as when tubercles or pneumonia coexist with the liquid effusion, the egophonic resonance of the voice is modified. It becomes louder and more forcible, and the key is not quite so high. It is in fact a mixture of egophony and of bronchophony. It resembles very much the voice of Punchinello, and it sometimes strikes the ear like the note of a trumpet.

I have now presented to you a summary of the most important physical signs of the healthy and of the diseased lung. Make yourselves, in the first place, perfectly familiar with the physical signs of a healthy lung. Practise upon each other repeatedly, deliberately, and when you have gained this object, turn to the wards of the Hospital, and study the signs of the diseased lung. Practice, attention, and a good ear will, with proper instruction, enable you to recognize these signs. But this is not enough: you must learn to connect these signs with the organic, the physical changes in the condition of the lungs which produce them. This must be done by a careful study of the post mortem appearances, which exist in connection with these signs. This is an effort of reason and of reflection. It elevates the art of percussion and auscultation into a science—a part of that medical science which all devoted lovers of their profession are laboring to improve.

LECTURE II.

RATIONAL AND CONSTITUTIONAL SYMPTOMS OF PULMONARY DISEASE.

The rational symptoms of pulmonary disease.—Pain, dyspnœa, cough, expectoration.—The constitutional symptoms.—Febrile symptoms, the pulse, emaciation, loss of strength, affections of remote organs.

The rational symptoms of pulmonary disease are few and simple in their character, and are all present in almost every case in which these organs are primarily affected by disease. They are sometimes, however, latent, and the chief cause of this latency will be found in the development of disease in some other organ, or in a general affection of the system, as for instance, fever. There are four rational symptoms of diseased lung, *pain*, *dyspnœa*, *cough*, *expectoration*. In the present lecture I shall pursue the same method I pursued in my lecture of yesterday. I shall describe, in a general way, the causes of these symptoms, and their influence in the diagnosis of pulmonary disease.

Pain is a very common symptom of pulmonary disease. It is frequently the effect of inflammation. In bronchitis, that is, in the early stage, it is a sensation of weight and of soreness under the sternum; in laryngitis, it is often accompanied by an uncomfortable sensation of heat and dryness. Sometimes it is accompanied, when the epiglottis is affected, by pain in swallowing. In simple pneumonia there is not usually much pain experienced, but rather an uncomfortable sensation of heat in the chest. It is in pleurisy that you will find the most marked pain to exist. This is usually, when severe, confined to a small spot under the nipple of the side affected. The more severe the pain, the less extended is it. The explanation of this is simple. The whole pleura is in a state of inflammation. The stretching of this inflamed membrane during inspiration causes pain, and that portion which is first acted upon, first reveals its morbid sensibility. This portion is of course the

point where the natural expansion of the parietes of the chest is most rapid and considerable, below and to the outside of the nipple. Often the pain produced by stretching this portion of the pleura is so severe, that the act of inspiration is at once arrested, while in cases in which the pain is less marked, it commences at this spot, and spreads more or less to the other portions of the affected side, as the chest continues to expand. Even where the pleurisy is a secondary affection, dependent upon some disease at the summit of the lung (pneumonia, tubercles), it is apt to be felt in the lower portion of the affected side from the tendency of the pleuritic inflammation to spread beyond its original seat, although in cases where it is mild in degree and limited, it reveals itself by pain in the spot where it is originally developed. Thus, in tuberculous disease of the lungs, you will often find a limited pleuritic pain at the summit of the lung, under the clavicle, or under the scapula.

One of the most important characteristics of pleuritic pain is its fixedness. But at the commencement of the inflammatory attack, it is, sometimes, wandering. I have known it to be first felt in the abdomen, but it soon locates itself in its proper seat.

Another very frequent kind of pain in pulmonary disease is *neuralgic* in its character. It is external, and has its seat in the intercostal nerves. Sometimes it is dependent upon a general neuralgic condition of the system, a condition not infrequent in feeble and delicate constitutions. When confined to the chest, it does not necessarily indicate pulmonary disease. I am satisfied that persons who have simply weak lungs are disposed to neuralgia of the intercostal nerves. It is, however, frequently a symptom of even severe pulmonary disease. In many cases of tuberculous disease, the most frequent pain in the chest is neuralgic in its character. In a case of cancer of the lung, to which I shall have occasion to call your attention, severe neuralgic pain, beginning in the arm, was the first symptom, and after death a cancerous mass was found irritating the nerves of Wrisberg.

The pain in bronchitis is dull, seated under the sternum, and not materially increased by coughing, or by a full inspiration. The pain in pleuritis is sharp, remarkably increased by a full

inspiration, seated under the nipple, fixed, limited in extent, without much external tenderness, and is attended by fever. The pain in the intercostal nerves is sometimes extensive, sometimes limited: wandering, intermittent, attended with external tenderness, especially at three points along the course of the nerve, where the superficial cutaneous branches are seated, viz., near the transverse process of the vertebra, in the lateral region, and about the junction of the rib with the sternum. This pain, when severe, is increased by a full inspiration and by coughing. Its chief distinctions from pleuritic pain are, its wandering, intermittent character, external tenderness, and the absence of febrile symptoms.

Dyspnœa is the effect of any cause which interferes with the action of the air on the blood in the capillaries of the air-cells. It is a sensation like hunger and thirst, which indicates a want that is not supplied, and is attended by increased labor and acceleration of the respiration. You will readily understand that conditions quite opposite in their nature may produce this result. Thus, the too rapid passage of the blood through the lungs, as after exercise, is the simplest cause of this symptom. The same thing happens in cases of general febrile excitement. In other cases, it is the retardation of the passage of the blood through the capillaries of the lungs, as occurs in heart-disease, and especially in mitral disease. Again, the source of the difficulty is in the lungs. The air cannot enter freely into the air-cells from compression of the lungs, as in pleuritic effusion, hydrothorax, pneumothorax, tubercles; or from obstruction of these cells, as in pneumonia; or from a thickened and rigid state of the air-cells, which may contain much air, but which acts imperfectly upon the blood in the capillaries, from being slowly displaced by fresh air, and from the thickened state of the cell-walls, as in emphysema. Finally, the cause of the imperfect action of the air on the blood, may exist in the larynx, the trachea, or the bronchi. These tubes may be compressed by external tumors, aneurism, enlarged lymphatic glands; or by internal tumors. Or a thickening of the lining membrane, or an increased secretion of mucus, or a spasmodic contraction of the tubes may exist—all causes operating more or less decidedly to prevent the admission of air into the lungs

These causes, so different in their nature and in their seat, all operate in producing the same result, an imperfect action of the inspired air upon the blood in the pulmonary capillaries. Several of these causes are often combined to increase the effect, either permanently, or for a time. Thus, the natural number of inspirations may be increased from sixteen or eighteen in a minute, to forty, or more, in the adult, and to sixty or eighty in children, and even far above this in frequency.

Dyspnœa, like pain, is not unfrequently a nervous symptom. Patients who are dyspeptic, frequently complain of it, and attempt to relieve the apparent want of air, by making from time to time a deep inspiration—by sighing. In these cases there is no apparent want of air in the lungs, nothing which indicates any unusual rapidity, or delay in the pulmonary circulation. It depends, very likely, upon the condition of the blood itself, or, more frequently, upon the pressure of the distended stomach. It would, probably, be impossible to find a case of simple pulmonary disease, unattended by *cough*. When, however, the disease is rendered latent, by an affection of some remote organ or of the general system, as in fever, cough may be entirely absent. This symptom may be trifling, or of rare occurrence, or violent and frequent in its development. It is sometimes paroxysmal, as in whooping-cough, and it is apt to occur at particular times in the day, in the morning after rising, as in tubercles of the lungs. You may sometimes form a pretty good idea of the disease by watching the character of the cough. Thus, if the disease is in the larynx, the cough is apt to be loud and barking; if in the bronchi, loud and ringing; if in the air-cells, or in the pleura, feeble and suppressed. As a general rule, a slight cough is more serious than a prominent one. The slight hacking cough of pulmonary tubercles in the early stage, the feeble, suppressed cough of pleurisy and of pneumonia, stand in very striking contrast, as symptoms of danger, with the loud ringing cough of bronchitis.

Cough is often a sympathetic symptom, when it is usually dry, and often trifling in degree, as in affections of the digestive organs.

The cough is sometimes dry, and continues so for a long time,

but sooner or later, it is usually attended by *expectoration*. This expectoration is commonly mucus from the bronchi—transparent, thin, and frothy; opake and thick, muco-purulent, or purulent. Sometimes it is streaked by blood, as in bronchitis; sometimes the blood is intimately mixed, so as to give the expectoration a rusty look, as in pneumonia; sometimes pure blood is expectorated, as in tubercles. A copious expectoration generally indicates an advanced stage of disease (bronchitis, tubercles, gangrene, pneumonia, pleurisy). It is sometimes offensive, sometimes sweet, or saline, to the taste.

Some forms of expectoration are diagnostic, as the rusty expectoration of pneumonia, the grayish, defluent, fetid expectoration of pulmonary gangrene, the expectoration of blood of tubercles. The expectoration of cretaceous masses is also an indication of tubercles, when the disease is tending to recovery. Occasionally, distinct masses of tubercles, opake and softening, are expectorated, and very frequently the greenish, opake, muco-purulent expectoration, when a tuberculous abscess exists in the lung, is streaked by yellowish lines, which are softened, broken down tuberculous matter.

Chemical and microscopic examinations of the expectoration, have not as yet discovered much that is valuable in a diagnostic point of view. You will find, under the microscope, epithelial scales from the mucous membrane of the bronchi, ordinary pus globules, and occasionally fatty matter and cholesterine, arising from a tuberculous abscess. A remarkable illustration, however, of the value of a microscopic examination in the diagnosis of hemorrhage from the lungs, has occurred to me during the past year. A young lady, with some suspicious chest symptoms, expectorated suddenly several ounces of a dark-red, or chocolate-colored fluid. This was suspected to be blood, in part at least. But on examination by the microscope, not a single blood-disk could be discovered, but an immense number of epithelial scales, from the mucous membrane of the bronchi, sufficient to give the fluid its reddish color.

All the inflammatory diseases of the lungs are attended by the *constitutional symptoms* of fever. In acute bronchitis, they are usually mild, and of short duration; in pneumonia and in pleurisy

they are much more severe, and of longer duration. In the former disease, febrile excitement seldom continues for a longer time than three or four days; in pneumonia, it usually continues a fortnight, and in pleurisy, often for a much longer period of time. In chronic diseases of the lungs (empyema, tubercles), the fever usually assumes the hectic form. Night-sweats, however, which are usually a prominent symptom of hectic fever, are not necessarily a symptom of disease. They sometimes are the effect of mere debility. Of course, when inflammation exists, the pulse is usually accelerated, often increasing from seventy-six in a minute, which is the standard of health in the adult, to one hundred and twenty pulsations. But an accelerated pulse is not necessarily an indication of inflammation. Deep-seated irritation in the lungs, as from the deposit of miliary tubercles, will often increase its frequency, permanently, to one hundred, or one hundred and twenty pulsations. So general is the excitement of the pulse, in the early stage of the tuberculous irritation, that, occasionally, when I have found it at about the natural standard, I have ascertained by inquiry that the patient had, naturally, a slow pulse, perhaps not more than sixty pulsations in a minute. In such a person, a pulse of seventy-six would be an accelerated pulse. Great differences also exist in the frequency of the pulse in pulmonary diseases, which are dependent upon age, and upon different degrees of nervous excitability. In old people, the pulse is less easily excited; in children, it is not only naturally more rapid than in adults, but is so easily excited, that its increased frequency in health probably depends, in part at least, upon the constant operation of some exciting cause. Thus, according to the observations of Ledeberder, the pulse at the moment of birth is only eighty-three in a minute, while in three or four minutes it rises to one hundred and sixty. It is important to appreciate the influence of early childhood on the frequency of the pulse, as you will thus be better able to appreciate the effect of disease. Valleix has given some valuable information on this subject, in his chapter on the clinical examination of children, and his observations are the more valuable, because he took the greatest pains to ascertain that the subjects to be examined were in good health, and to

guard against the causes of arterial excitement. He found the mean number of pulsations during the first ten days after birth, the child being perfectly quiet and healthy, was eighty-seven, the maximum being one hundred and four, the minimum seventy-six. In older children, probably from the more permanent influence of exciting causes, the frequency of the pulse is greater. Thus in children whose mean age was seventeen months, the mean number of pulsations was one hundred and twenty-four; in children whose mean age was sixty-four months—five to six years—the mean number of pulsations was one hundred and eight.

But the influence of a highly excitable nervous temperament, is not only observed in children, but frequently also in adults, and especially in females. The mere excitement of seeing a physician for the first time, will frequently accelerate the pulse ten or twenty pulsations in a minute. You must always remember this, in examining the pulse as an indication of disease. The different qualities of the pulse are more difficult to appreciate. A hard, incompressible pulse has been supposed to be more particularly connected with serous inflammations; a soft, full pulse, with inflammation of the substance of organs, or of mucous membranes; a quick pulse, with nervous irritability.

Emaciation is a very important symptom in chronic pulmonary disease, for it sometimes possesses much diagnostic value, especially if it exists without any dyspeptic complication. It is particularly valuable in the diagnosis of incipient phthisis, of which it is often the first symptom. It not unfrequently happens, that patients lose flesh, without apparent cause, for a considerable period of time, long before any local symptoms have occurred. In almost all cases, it is a very early and prominent symptom of this condition of the lungs, and serves to distinguish it from the two chronic diseases with which it may be most easily confounded, chronic bronchitis, and emphysema. Of course, in all acute inflammatory affections of the lungs, a certain degree of temporary emaciation occurs, which is speedily removed when the disease disappears.

Loss of strength is a usual attendant on emaciation.

Severe pulmonary disease is sometimes *latent*, that is to say, it is unattended by any rational or constitutional symptoms, or these

symptoms are but few in number, and indistinct. In febrile diseases, as in typhoid or typhus fever, in the eruptive fevers, acute inflammation may attack the lungs without being attended by pain, or by cough. Dyspnœa is less frequently absent, but this may be a symptom of the general febrile excitement. If, however, it exists in a more marked form than is usual in such cases, or if it has increased without apparent cause, the physical condition of the lungs should be carefully examined. Generally, the true condition of the lungs is at once revealed by this examination. The physical signs of bronchitis, of pneumonia, or of pleurisy, are easily discovered.

The same latent tendency exists in chronic cases, and from the same cause—the pre-existence of disease in some remote organ, or its subsequent development. A medical gentleman died in this city during the past year who had suffered for a long time with symptoms of disease of the urinary organs and of the rectum. This disease was found, after a post-mortem examination, to be a cancerous affection of these organs: at the same time the lungs were found full of cancerous deposits. Yet neither the patient, nor the intelligent physicians who attended the case, ever suspected any pulmonary disease. I remember the case of a young lady, whom I attended many years ago, which made a great impression on me. She was suffering from pain in the head and from chronic diarrhœa. She emaciated rapidly, had hectic fever, but never any symptoms of pulmonary disease. No cough, or pain in the chest existed, and the respiration was easy and natural; yet, after death, the lungs were found full of tubercles beginning to soften. My subsequent experience has presented many analogous cases, the true character of which has been disclosed by a physical examination of the chest.

You may ask the question, Which of the three classes of symptoms, or signs, which I have indicated, are the most important in the diagnosis of pulmonary disease? I answer that they are of equal importance in diagnosis. No case should be considered as positive, until each of these classes of symptoms has been carefully examined, and carefully compared with each other. In cases in which they are not all well marked, the diagnosis must be regarded as more or less doubtful, although, even

then, the deficiency in one class of symptoms may be admirably counterbalanced by the distinctness of another class.

The difficulties attending the thorough investigation of pulmonary diseases are by no means trifling. Many mistakes are made by the carelessness or ignorance of the physician. The art of examining a patient implies many high qualifications: a natural talent for observation improved by practice, an ardent desire to learn the truth, an accurate knowledge of other diseases which may be mistaken for the disease under examination. Many difficulties also arise from the ignorance, carelessness, or stupidity of patients. This is especially true in relation to Hospital patients. They are often surprisingly ignorant of their constitutional tendencies to disease, of their previous diseases, of the time when their actual symptoms commenced, and of their subsequent development. All these unfavorable circumstances render the art of diagnosis a work of much labor, of many disappointments. Still persevere. An intelligent mind, a sincere love of truth will gradually overcome these difficulties, and the reward will be a knowledge of the diseases of your patients, and of the proper indications of treatment.

LECTURE III.

BRONCHITIS.

Pathological anatomy.—Obliteration and dilatation of the bronchi.—Cirrhosis of the lung.—Causes.—Primary and secondary bronchitis.—Acute and chronic bronchitis.—Symptoms of the different forms of the disease.

The air-passages are lined by a smooth, white, transparent, and delicate membrane which adheres, for the most part, very closely to the subjacent tissue. This membrane is very frequently affected by inflammation, and the disease has received different names according to the particular portion of the membrane that is affected. Thus we have laryngitis, tracheitis, and, finally, bronchitis. It is only when the inflammation extends to the air-

vesicles that it is called pneumonia. The subject of the present lecture is bronchitis.

It is not my intention to enter upon a minute description of the bronchi. I shall simply allude to one or two facts in their anatomy which are of importance in a pathological point of view. In the first place, the bronchi are provided with a coat of circular muscular fibres, surrounding the tube probably in the smaller divisions, and capable of diminishing very materially the calibre of the tube. Secondly, the membrane which lines these tubes gradually assumes more and more the characters of a serous membrane as it descends to the smaller branches, until, reaching the air-cells, it becomes quite serous in its character. This fact is not capable of a strict anatomical demonstration, but I am compelled to admit it to explain the pathological facts you will be constantly called to observe.

As a general rule, in the examination of cases of bronchitis after death, you will not find very strongly marked evidences of inflammatory action. The evidences you will find are these: the mucous membrane presents more or less redness, sometimes continuous, more frequently appearing in spots varying in size—in patches of considerable extent, in minute specks or dots—and composed of blood-vessels, short and tortuous and running together, or broken up into mere points. Sometimes you will notice a diffused redness without the distinct appearance of vessels; at other times, more decided spots of ecchymosis. In the acute form of bronchitis, this redness is more or less bright in its hue, approaching vermilion in color. In some cases, however, it is of a darker hue, especially when the disease has existed for a considerable time. Indeed, in the chronic form, the redness will frequently assume a violet tint, or even a slaty hue.

In most cases of bronchitis, the mucous membrane has lost more or less of its natural polish and transparency; still these changes are not usually very marked, especially in the acute form. So that when the lung itself is congested by blood, a little care is necessary not to mistake the redness of the pulmonary tissues, as seen through the transparent bronchus, for a redness of the bronchus itself. Indeed, if you examine a case of bronchitis with sufficient care, you will perceive that the redness is

not in the mucous membrane itself, but in the subjacent cellular tissue. It is here that the capillary vessels ramify; and in inflammation they are distinctly seen to be situated beneath the mucous membrane, appearing, as I have already stated, in the form of small tortuous lines, more frequently broken up into dots and points, than running together so as to form an imperfect arborescence. When you notice a predominance of the arborescent injection, you may, generally I think, refer it to a passive or venous congestion, having its seat in the larger branches, although it may happen that the capillary or inflammatory injection will extend to arterial branches of a considerable size, and remain in them after death. But this I think, at least to any extent, is of rare occurrence. This difference in the form of the injection is of importance, because it possesses a diagnostic value in estimating the changes arising from diseased action.

It sometimes happens that the mucous membrane lining the bronchi is more friable than natural, and more easily detached from the subjacent parts. It may also appear somewhat thickened. These changes, more apparent, particularly the latter, in the chronic form of the disease, can only be distinctly recognized in the larger tubes, which can be readily opened and submitted to the test of the scalpel. In the smaller tubes, all that you can do is to cut them across and observe the divided extremities, when more or less redness of their mucous surface may be observed, together with some turgescence. You can commonly form the most satisfactory estimate of their inflamed condition by observing their contents as they exude by pressure from the divided tubes.

The first effect of inflammation on the mucous membrane of the air-passages, is to stop secretion—the parts are tumid and reddened, but they are dry. This condition is transient. Soon an increasing discharge of thin transparent mucus takes place, which becomes abundantly aerated from the act of coughing, and which gradually becomes more viscid and consequently less aerated, opake, and yellow, from its approach to the characters of pus. This mucous, or muco-purulent secretion, is almost always found in more or less abundance in our examinations after death. It may be so abundant as to prevent the usual col-

lapse of the lungs; it may fill the smaller tubes, or be found only in the larger bronchi. In other cases, you will find lymph effused, especially in the smaller bronchi, and the frequency with which this is found, especially in the capillary bronchitis of children, proves the analogy of the membrane which lines these tubes to the serous tissue.

Indeed, the frequency with which the bronchi are *obliterated* by the effusion of lymph, would be much more serious in its consequences, were it not confined chiefly to the smaller tubes, for the consequence is atrophy of the air-cells supplied by the tube. We sometimes meet with obstruction to the larger tubes, but usually from a different cause, from a partial thickening of the walls of the tube producing a true stricture. A tumor, as an enlarged gland, an aneurism, may also obstruct the larger bronchi, and, by interrupting the function of a considerable portion of the lung, induce more or less dyspnœa.

Dilatation of the bronchi is another consequence of inflammation. It may be general or partial. In some cases, you will find the tubes supplying a whole lung, or a lobe, dilated, except the bronchi of the first and second order, which are seldom or never affected. In this case, the bronchial tubes when slit open by the scissors may be as large, or larger, than the trunk from which they originate, and are easily exposed to the very periphery of the lung, where they terminate in cul-de-sac. The appearance thus presented recalls the appearance of the fingers of a glove slit open. In other cases, the tubes are abruptly enlarged at a particular point—a single tube, or several tubes near each other uniting to form an irregular cavity. In other cases, you will find a succession of dilatations in the same tube which has received the name of fusiform dilatation. Finally, the termini of the bronchi are alone dilated, forming small rounded cavities capable of holding perhaps a small pea.

The condition of these dilated tubes varies very much in different cases. Sometimes, especially where the dilatation is general, you will find the tubes thickened as well as enlarged—the fibrous coat is more distinct and firmer than natural, and the mucous membrane thickened, softened, and more or less rough. In other cases, especially where the dilatation is partial and limited, as

in the saculated and fusiform varieties, the coats of the dilated tubes are even thinner than natural, and the mucous membrane, although sometimes more red than natural, is usually pale, smooth, and transparent as in its healthy condition. In the first-named condition of the bronchi, the bronchial mucous membrane is in a high state of chronic inflammation, as is also indicated by a more or less abundant secretion of thick purulent mucus. The same evidence of high inflammatory action is observed in the terminal dilatations of the bronchi, a form of dilatation which you will especially notice in children; for in these cases the cavities thus formed contain a distinct puriform fluid. But in other cases, the evidences of inflammation of the bronchial mucous membrane are not so apparent, and if they exist at all, exist in a much less degree. This is especially the case in those forms of partial dilatation accompanied by a thinning of the walls, in which the mucous membrane retains its natural transparency, is but little if at all softened, and in which the bronchial mucus is but moderately opake, or is even transparent.

Here are evidently conditions very different in these different cases. If you look beyond the bronchi to the lung supplied by them, you will find an equal difference. In some cases, the lung appears to be simply compressed by the neighboring dilatations, resembling in appearance a portion of lung compressed by a serous effusion into the pleural sac; this is especially noticed in cases in which the dilatation is general, and in which the tubes themselves exhibit a high degree of inflammation. It would appear, in these cases, that the primary cause of the dilatation is the loss of elasticity in the bronchi from inflammation, and that the forcible inspirations of coughing and the accumulation of mucus lead directly to the dilatation of the tubes, and to the consequent pressure and condensation of the surrounding pulmonary tissues; while in other cases, especially in the partial dilatations and where the walls are thinned, the mechanism of the dilatation is apparently different—its starting point being, not in the tubes themselves, but in the lung. In extreme cases, we find the structure of the lung itself remarkably altered. Thus, in a case reported by Laennec, the whole lung, not larger than the two fists, was transformed into a substance, apparently between

the cartilaginous and the fibrous tissues. The upper lobe, closely united to the lower, was of a grayish slate color, while the latter was as white as a tendon. When cut into thin slices it was slightly transparent, and had nothing of the flaccidity of a lung simply compressed—the bronchi were evidently dilated from their first to their last divisions, which terminated in cul-de-sac—the smallest branches were obliterated. The mucous membrane lining the dilated tubes was of a dark-red color, slightly thickened, and covered by a thick opake mucus resembling, somewhat, soft cheese. In such a case, the first step in the series of morbid changes was not, probably, in the bronchi, but in the intervesicular cellular tissue of the lung—a slow inflammation attacking this tissue, followed by a deposit of lymph and by subsequent contraction leading to an atrophy of the whole lung, except the bronchial tubes, which enlarged to fill the space left by the shrinking lung, aided no doubt by the efforts of coughing. Or it may be, that the bronchi are first inflamed, and the inflammation is propagated from them to the cellular tissue, and then the same results follow, viz., contraction of the lung, and subsequent dilatation of the tubes. This view of the subject was first presented to the profession by Dr. Corrigan of Dublin; and from the mechanism of the organic changes being somewhat similar to those inducing cirrhosis of the liver, the affection was called by him, *cirrhosis of the lung.*

Rokitanski has taken a different view of the subject, which may explain may cases, but I think not all. In his opinion, the first step is a capillary bronchitis, which obliterates the tubes by the effusion of lymph. Then the pulmonary tissue supplied by these tubes becomes atrophied, contracted, whitish, or dark from the deposit of black pulmonary matter, and the dilated bronchi enlarge to supply the vacant space. It is probable that all these modes of dilatation may occur, either singly or united. One important fact gained by such investigations is this: that the pulmonary structure is often seriously implicated independently of the bronchial dilatation, and that these changes tend to interfere with the proper oxygenation of the blood, not only by obliterating the air-cells, but also by producing serious obstruction to the pulmonary circulation.

Inflammation of the bronchi is most frequently double, that is, it affects both lungs and generally the lower lobes. It may be general, but it is very rarely, if ever, found in the upper lobes only.

Bronchitis is either a primary or a secondary affection. In the primary acute form its chief exciting cause is exposure to wet and cold. In our variable climate, and especially in the spring season, there are few individuals who have not experienced repeated attacks. This tendency exists in all classes, although certain constitutions are more disposed to it than others. There are many individuals in whom wetting the feet, exposure to a draught of cold damp air, or to any other cause of sudden chilliness, is almost sure to induce an attack. In these cases the frequent repetition of the attack is apt to lead to the chronic form of the disease.

There is one form of bronchitis, whooping-cough, which recognizes a specific cause, probably a morbid poison acting directly on the bronchi. Direct local irritation may also produce bronchitis, as the inhalation of gases and of dust, especially of metallic dust. Certain substances, in particular constitutions, produce the same result. Thus, new-mowed hay, in some persons, will induce an attack often attended with marked dyspnœa. I have known ipecacuanha in powder produce the same consequences. The flowering of roses, in some persons, will spoil the month of June, and I have seen individuals who habitually lose the enjoyment of our fine autumn from similar attacks, but from what particular cause I have not been able to determine. The influence of certain general causes, atmospheric perhaps, in causing, and more particularly in prolonging bronchitis, are curious and would be instructive, if we could understand their *modus operandi*. There are many persons who cannot visit certain localities, and these sometimes very circumscribed in extent, without experiencing a high degree of bronchial irritation. A change of locality will sometimes bring a protracted case, which has resisted all the efforts of art, to a suddenly favorable termination. A change to the city will sometimes benefit the resident in the country, and *vice versa*.

Bronchitis, both acute and chronic, is frequently a *secondary*

affection. I do not remember any disease of the lungs in which it does not play a more or less prominent part. Thus, it constantly occurs in tubercular disease, in cancer, in emphysema, and in the acute inflammations, as pneumonia and pleurisy. It is, also, very frequently met with in those general affections caused by the influence of a morbid poison in the blood, as in continued fever, the eruptive fevers, and in the influenza. There are also frequent attacks of bronchitis, which depend on some remote irritation, and especially on gastric or intestinal disturbance, a fact which should be carefully remembered. It is also of common occurrence in heart disease.

The influence of certain trades and professions in producing bronchitis has excited much attention in England, especially in the large manufacturing towns. It is to Mr. Thackrah, to Dr. Knight of Sheffield, and to Dr. Darwall of Birmingham, that we are chiefly indebted for the information we possess on this subject. These observers have divided those peculiarly subject to bronchitis into three classes. First, those who suffer from the inhalation of dust. Of these, those who inhale metallic dust suffer most severely; as the needle and edge-tool makers, the gun-barrel grinders. In England, this affection is vulgarly known as the pointers' cough, the grinders' asthma, or rot. This is the most severe form of the disease; indeed, it is frequently fatal. Those who inhale a vegetable or animal dust suffer less; as sawyers, millers, starch-makers, flax-dressers. In the second class, are placed those who are exposed to sudden changes of temperature; as glass-blowers, bakers, brewers, brass and iron founders, and the like. Finally, in the third class we find those in whom bronchitis is induced by sedentary habits; as tailors, clerks, shoemakers, jewellers. I have mentioned these causes of bronchitis in the order of their frequency, and of their importance to life. The pointers' cough appears to be a very frequent cause of death, inducing ulceration of the mucous membrane, and disorganization of the lungs. It is said that the magnet has been successfully applied to remove or to mitigate this evil.

Drs. Graves and Stokes of Dublin, have described a form of secondary bronchitis associated with constitutional syphilis,

which assumes a grave character from its resemblance to tubercular phthisis. It is, however, accompanied by secondary symptoms, and often yields spontaneously after the appearance of a cutaneous eruption.

A dry asthmatic bronchitis also occurs in gouty subjects, with a sibilant or sonorous rhonchus, which frequently disappears when the joints become affected.

I have seen a form of bronchitis attended by a dry, irritable cough, by irregular chills followed by febrile excitement, and perhaps by night-sweats, after exposure to the malarious influence, and which yielded readily to the arsenical solution.

Bronchitis, in its primary acute form, is seldom a fatal disease.* In most cases, indeed, it is so mild as hardly to require the interference of medical aid. In children, however, in whom it is apt to be capillary and general, it sometimes assumes a formidable aspect; also, in very old persons, in whom it is also capillary, and attended by a copious secretion of a thin serous fluid. Primary chronic bronchitis is also usually a mild disease. There are many persons who live in our climate, especially those somewhat advanced in life, who have a bronchitis for years without any serious interference with the general health. When, however, it is complicated by dilatation of the bronchi, it assumes a more grave aspect, and death may follow with most of the symptoms of tuberculous disease.

Secondary bronchitis may be a serious complication. When it occurs in connection with acute diseases, it may render the prognosis much more unfavorable. This is frequently the case in continued fever, and in the eruptive fevers. Occurring in cases, in which the circulation of the blood has been long impeded, as in emphysema, or in heart disease, it aggravates greatly the

* In the City Inspector's Report for 1851, there are 132 deaths from bronchitis, in a general mortality of 16,978, in which 924 died of pneumonia, and 30 of pleurisy. Ninety-two of the cases of bronchitis died between December and June; forty between June and December. Eighty-four were males, forty-eight were females. The proportion, according to age, was the following: 1 year, 34; 1 to 2 years, 17; 2 to 5 years, 18; 5 to 10 years, 7; 10 to 20 years, 3; 20 to 30 years, 8; 30 to 40 years, 8; 40 to 50 years, 5; 50 to 60 years, 12; 60 to 70 years, 5; 70 to 80 years 9; over 80 years, 5.

dyspnœa, and assumes a character which has been called *suffocative.* While, as a symptom of remote irritation, especially of gastric derangement, although trifling in degree, it may excite much apprehension from the idea that it may be connected with a tuberculous affection of the lungs.

Laennec has described several forms of bronchitis, which are not now usually recognized as distinct varieties of this disease. His *pituitous catarrh*, in which the expectoration does not pass beyond the stage of simple transparent mucus, however abundant it may be, is noticed in œdema of the lungs, and in the pneumonia notha of old persons, in the more acute cases. It is also associated with tubercles in their earlier stage. The *dry catarrh* of the same author is also usually a secondary affection. It occurs in emphysema, in tubercles, and especially in the forms of secondary bronchitis, dependent upon remote or upon constitutional irritation. It is the form dependent upon gastric or intestinal irritation, upon hysteria, upon the gouty diathesis. In this dry catarrh, the anatomical characteristic is described as a swelling of the mucous membrane of the smaller bronchi, accompanied by a trifling secretion of very viscid mucus, sometimes appearing as an expectoration of a whitish, semi-opake, pearly matter. On examining the chest, you will find the sibilant rhonchus, and the respiratory murmur feeble in certain portions where the bronchial obstruction is considerable, and liable to become distinct again suddenly, especially after coughing. No doubt a spasmodic contraction of the tubes, in many cases, is the cause both of the temporary feebleness of the respiration, and of the sibilant rhonchus. In these cases, there is also more or less dyspnœa, which may be aggravated to a paroxysm of asthma, if an acute attack of bronchitis supervene. Laennec regards this dry catarrh as the forerunner of emphysema. It is certainly very often associated with it.

Acute primary bronchitis commences as an inflammation of the nasal fossæ, with a sensation of heat, dryness, fulness in the nose, speedily followed by a copious discharge of thin, transparent mucus. The inflammation extends to the throat, producing dysphagia, and a degree of redness and swelling of the fauces; to the larynx, producing hoarseness, and finally to the

bronchi, producing cough and dyspnœa. It is not uncommon, however, for the attack to commence in the larynx, or in the bronchi. It is usually ushered in by a moderate degree of constitutional disturbance; a slight chilliness, with aching in the limbs and in the head, followed by heat of the skin, impaired appetite, diminution of the secretions, and acceleration of the pulse. The cough in this early stage is frequent, dry, hoarse, and sonorous, there is some dyspnœa, and a sensation of weight and of soreness along the sternum. If you examine the chest, you will not probably discover any thing abnormal, except, perhaps, a little harshness of the respiratory murmur, or a sibilant or sonorous rhonchus, especially over the posterior portions of the lungs. Soon, however, a more or less abundant secretion of mucus occurs, which begins to be expectorated, at first with difficulty, as a thin, transparent, frothy matter, frequently streaked by blood.

About the fourth day after the attack, the expectoration gradually undergoes a change—opake yellowish streaks appear, which become rapidly more numerous. It becomes more abundant, less frothy, less streaked by blood, and, at the same time, the cough becomes more easy, the dyspnœa and soreness of the chest diminish, and the constitutional symptoms subside. The fever abates, the appetite improves, the secretions increase. About the tenth day, the whole expectoration has nearly changed its character, and has become opake and yellowish, but with some remains, perhaps, of the thin, transparent secretion. If you now examine the chest, you will find that the harshness of the respiratory murmur and the rhonchi have nearly or quite disappeared. In most cases, indeed, you can detect nothing abnormal in the physical condition of the chest, although in the more severe cases, a mucous rattle may be heard, *and always at the base of each lung.* In the progress of the case, this also disappears, the expectoration gradually diminishes, the cough ceases, and perfect restoration ensues after a period of three or four weeks. In no case, however severe the attack, is there any dulness discovered on percussion.

Such is the history of an ordinary, but well-developed case of acute primary bronchitis in the adult. A mild form of disease,

free from danger unless neglected, running a course not definite in its duration, yet ceasing within certain limits. But it is not always this mild disease. In young children especially, it often affects the bronchi throughout the lungs, and especially the capillary divisions. In such cases, the constitutional disturbance is much more decided, the dyspnœa is more severe, and the physical signs more distinct. The disease may assume a suffocative character, and the high degree of fever may lead to the supposition that pneumonia is present. But this is not necessarily the case. The more minute bronchi are affected, which are lined by a membrane approaching the serous membrane in its character, and in serous inflammations the constitutional disturbance is always of a higher grade. The effusion of lymph into the tubes and the general diffusion of the disease through both lungs explain the aggravated dyspnœa, while the susceptibility of all young subjects to morbid impressions sufficiently explains, to the careful observer, why the bronchitis of children sometimes assumes so grave an aspect. In the physical examination of such cases you will still find that there is an abundance of air in the lungs. There is no dulness on percussion, but the respiratory murmur, feeble, perhaps, is everywhere marked by a sibilant or by a sonorous rhonchus, which gradually changes into a mucous rattle, especially at the base of the lungs. The symptoms, however, gradually subside by appropriate treatment, or they may assume an aggravated and fatal tendency. The dyspnœa increases (sixty to eighty inspirations a minute), and becomes noisy, the alæ nasi are much distended, the countenance anxious, and alternately pale and livid after coughing, the pulse rapid, feeble, and irregular, and finally delirium may ensue, with great restlessness.

In old people the disease is also, sometimes, an aggravated attack. It is in this case also capillary, and attended by a remarkable secretion of serum; it is the peripneumonia notha of the old writers. It is marked by much dyspnœa, obscure pain in the chest, a distressing cough attended by a free watery expectoration, moderate febrile excitement, a tendency to coma, and to a typhoid condition. Such cases frequently prove fatal, and on post-mortem examination you will find the lungs exhibit-

ing, indeed, but few traces of inflammatory action, but discharging from the bronchi and air-cells an abundance of transparent frothy serum. It is, indeed, an acute form of œdema of the lung, called by Cruvelhier pneumonia œdematodes. It is not unusual, however, for both these forms of acute capillary bronchitis, at the two extremes of life, to pass into pneumonia.

Chronic primary bronchitis is also usually a mild form of disease. It is not uncommon for individuals advanced in life to cough and expectorate every day for years, especially after rising in the morning, and yet continue to enjoy very tolerable health. They may suffer somewhat from dyspnœa, particularly when the disease is aggravated by exposure, but this is seldom more than a trifling inconvenience. Under such circumstances also they may lose a little flesh, but this is soon regained. They may present a mucous rattle at the base of the lungs, or this may be absent. But when dilatation of the bronchi supervenes the case is different, especially if the dilatation is considerable. A more intense degree of inflammation, a more purulent secretion often exists, and the constitution suffers. Indeed, well-developed hectic fever may supervene with its train of attendants, emaciation, loss of strength, and weakness of the digestive functions, in fact, all the constitutional symptoms of advanced tubercular disease may be present. The functions of the lungs also suffer more when this complication exists, for the pulmonary tissues are more or less compressed or even altered in their structure, and dyspnœa from imperfect æration of the blood ensues, or dropsical effusion, or perhaps hemopthisis from obstruction to the pulmonary circulation. The physical signs also undergo a change. If the dilatation be considerable you will discover that there is a perceptible dulness on percussion, and a bronchial respiration masked more or less by a mucous rattle. You will also perceive an increased resonance of the voice. In an aggravated case, the diagnosis from tubercular disease is by no means easy. In the two cases, the same constitutional symptoms may exist, also the same rational symptoms, at least in many particulars, while the physical signs revealing evidence of pulmonary obstruction, and even of a cavity in the lungs and a falling in of the walls of the chest when the lung is contracted or

atrophied, might easily confirm you in error. The chief points in the diagnosis of dilatation of the bronchi from tuberculous disease are these: the advanced age of the patient, the absence of hemopthisis (although this symptom has been remarked in this form of bronchitis), and of tuberculous matter in the expectoration, the diffusion of the dulness on percussion and of the bronchial respiration, existing sometimes over a considerable extent of both lungs. None of these conditions are strictly diagnostic, and if, as may happen, the dilatation is limited, confined to the summit of the lung, the case would infallibly be regarded as tuberculous, and ninety-nine times in a hundred the diagnosis would be correct—for dilatation of the bronchi, as compared with tuberculous disease, is a very rare affection. I shall discuss this subject again when I speak, on some future occasion, of tuberculous phthisis.

When chronic bronchitis complicates other diseases, especially those of the pulmonary tissues, it differs in many important respects from the primary form I have just described. Where obstruction to the pulmonary circulation exists, as in emphysema, and in heart disease, congestion of the bronchi ensues, and with this, increased irritability inducing spasm. There is a striking feature in these forms of secondary bronchitis. It is aggravated dyspnœa. It modifies also the physical signs. The rhonchi are also much more diffused and more permanent in these cases. When I find a permanent rhonchus all over the chest, I am inclined to look beyond the bronchitis, and I expect to find some permanent obstruction either in the lungs or in the heart. It is in these cases also, when aggravated by an acute attack, that an abundant mucous rattle sometimes exists over the posterior, and especially the inferior portions of both lungs. This is the suffocative catarrh of Laennec, which, if you except the capillary bronchitis of children and of old people, is, perhaps, always a secondary bronchitis.

LECTURE IV.

BRONCHITIS.

Whooping-cough: its complications.—Application of the rational and physical signs of bronchitis.—Diagnosis.—Treatment.

THERE is a form of chronic bronchitis recognizing a specific origin, contagious, seldom attacking the same individual more than once, definite in its duration, which is known as pertussis, or whooping-cough. Like other forms of bronchitis, it is seldom a dangerous affection, although it may become so, as well as be prolonged much beyond its usual period by unfavorable circumstances. It is a disease of childhood, although you will occasionally see the middle-aged, or even old people, affected by it. Those who die with the disease usually present some complication which is the cause of the fatal issue, as pneumonia, pleurisy, œdema of the lungs, congestion of the brain, or some chronic disease which has gradually exhausted the vital powers. Like other affections which owe their existence to a specific poison, the anatomical traces of its existence are usually indistinct, and quite inadequate to account for the symptoms. Thus by post-mortem examination, you may find nothing beyond the ordinary appearances of bronchitis,—more or less injection of the bronchi, and an accumulation of mucus. Sometimes dilatation of the tubes is present and enlargement of the bronchial glands. If, in addition to these conditions, pneumonia, or pleurisy, or œdema of the lungs are present, the cause of death is more apparent. But you will often be compelled to look beyond the chest to find an adequate cause of death. You may find the vessels of the brain turgid with blood, effusion of serum into the ventricles, or beneath the arachnoid, effusions even of blood. Or the digestive organs may present the evidences of chronic disease—ulceration of the mucous membrane of the intestines, tuberculous deposits, which, although having an influence on the fatal issue, are in no way essential to the disease.

Whooping-cough, although its origin is specific, and it may occur at all seasons and under a great variety of circumstances, is not entirely independent of the ordinary influences which induce bronchitis. It is apt to be most severe and most difficult of management during the spring season, when bronchial affections are most prevalent. Indeed, it is quite apparent that the ordinary causes of bronchitis, although they do not induce it, at least aggravate it, prolong it, and render the occurrence of secondary affections more frequent.

It is difficult to fix with precision the time that elapses after exposure before the first symptoms appear. In most cases, indeed, there is no evidence of direct exposure. In other cases, the poison which causes the disease may remain latent in the system until developed by some accidental cause, as exposure to cold. Perhaps ten days, or a fortnight, may be mentioned as the usual time of its appearance after evident exposure.

The attack commences like an ordinary attack of bronchitis. The child coughs, is rather fretful, and perhaps feverish; the appetite is impaired. These symptoms may be very trifling, or they may disappear, except the cough, which continues dry and sonorous. After an interval of about two weeks, the true nature of the disease becomes apparent by the characteristic whoop, and the paroxysm is established. A series of rapid expirations ensue, which go on until the child seems in danger of suffocation. The face becomes red and swollen, the veins of the neck and face turgid, the eyes suffused by tears, until at length a long, sonorous inspiration follows, *the whoop*, and momentary relief ensues. But the same symptoms are rapidly repeated, until, at length, an expectoration of stringy, transparent mucus takes place, accompanied by the contents of the stomach, and the paroxysm is at an end. The child, panting, exhausted, often frightened by its violence, soon recovers from its effects, and returns to its sports as if nothing had happened. These paroxysms may be repeated, with more or less violence, every fifteen minutes, or only two or three times during the day. They may be so mild as hardly to attract attention after they have become familiar by repetition, or they may assume a fearful violence. The blood may gush from the nose, or even from the eyes and

ears during its continuance, or a more or less permanent chemosis becomes established.

If you examine the chest during the paroxysm, you will find nothing remarkable, only that the sound of the characteristic whoop does not pass into the lungs, but seems to stop high up, probably in the larynx, or in the trachea. If you search for signs during the interval of the paroxysm, you will still find but little to attract attention—a slight rhonchus, or a mucous rattle at the base of the lungs, may be heard, but even these are frequently absent.

The paroxysms usually continue, with different degrees of intensity, for about four weeks; they then gradually disappear, and the cough reassumes the character of that of an ordinary bronchitis, which may continue for two weeks longer.

Thus, the ordinary duration of an attack of whooping-cough is about two months: but it may continue for a much longer period. After the characteristic whoop has entirely ceased, even for one or two weeks, an attack of bronchitis may bring it back again; and this may be repeated until the disease assumes a chronic character. A delicate constitution may also dispose to a continuance of the disease, establishing a kind of habit which invigorating measures will alone remove.

There are two complications which are particularly to be feared in the progress of this disease. When you observe the paroxysm, and notice the flushed and swollen countenance, the turgid jugular veins, the chemosis, the gush of blood from the nose, and remember how predisposed children are to congestion of the brain, you will not wonder that this accident and its serious consequences should sometimes occur. A tendency to this complication may be suspected, when you find the face habitually flushed, the head hot, the child drowsy and indisposed to play, preferring to rest its head upon the mother's lap, restless in its sleep, moaning, and grinding the teeth. If these warning symptoms are allowed to continue, a frightful attack of convulsions may ensue, followed by coma and paralysis, and a speedy death.

Again, if you find the child feverish, and the cough suppressed and painful, if the pulse is excited, and the dyspnœa

[illegible] with the same listlessness, loss of appetite, and in- [illegible] and uneasy sleep at night, you must examine [illegible] pneumonia or pleurisy is forming there. On ex- [illegible] may detect dulness on percussion, bronchial [illegible] and other unequivocal evidences of pulmonary ob- [illegible] and the child may die in a few days with these for- [illegible] symptoms. It is worthy of particular notice, that [illegible] complications occur, either in the head or in the [illegible] may very much diminish in intensity, or en- [illegible] and thus lead a careless observer to suppose [illegible] was improving, while, in truth, the apparent re- [illegible].

[illegible] of the chest in most cases of bronchitis, estab- [illegible] of physical signs rather than their existence. [illegible] bronchitis, however severe, there is always [illegible] clearness on percussion. In some of the compli- [illegible] in emphysema of the lungs, the sound on percus- [illegible] unnaturally clear. In many cases, even when the [illegible] a suffocative character, the same clearness on [illegible] may be noticed; when a principal bronchus is ob- [illegible] time, dulness does not exist. The truth is, that, [illegible] dyspnœa, there is air enough in the cells; the [illegible] does not escape from them readily during the [illegible] ration, and is not so rapidly changed as the [illegible] require. When, therefore, you find the [illegible] on percussion, you may be certain that some- [illegible] bronchitis is present. If it be slight, limited [illegible] situated under the clavicles, it probably depends [illegible] deposits in the lungs. If it be more extended, it [illegible] on the existence of dilatation of the bronchi, [illegible] oedema, on pneumonia, on pleurisy, or hydrotho- [illegible] fact, the absence of dulness on percussion, is [illegible] value, both in the diagnosis and the prog- [illegible] disease. A chest that sounds clearly on [illegible] in a very serious condition, unless, indeed, [illegible] ant, as in emphysema, or in pneumothorax. [illegible] physical signs in bronchitis, is still further illus-

trated by the practice of auscultation. In simple cases, you will seldom detect any decided alteration in the natural respiratory sounds. In the early stage of the disease, the respiratory murmur may be a little harsh, and an occasional sibilant or sonorous rhonchus may be detected; obstruction of the bronchi, from mucus, or from spasmodic contraction, may render the respiratory murmur temporarily feeble in the portions of lung affected by the disease: but this transient effect usually disappears after coughing, to return again, perhaps, after a short time. As the expectoration becomes more abundant, these physical signs usually disappear. In the more severe cases, a mucous rattle may be heard at the base of the lungs—*always at the base of both lungs.* It is in the complicated cases, in secondary bronchitis especially, when connected with long-continued obstruction to the pulmonary circulation, as in emphysema, and in heart disease, that you will find the best-marked physical signs of bronchitis. In these cases, during an acute attack, the rhonchi are apt to be heard abundantly all over the chest, and to continue for a long time. It is in these cases, also, that the mucous rattle is most apt to be heard, after the attack has continued for a time. The obstruction to the circulation in these cases, seems to impress a peculiar irritability on the bronchi, by which the tendency to spasmodic contraction is materially increased; hence the development of the rhonchi. The same congestion seems also to favor the secretion of mucus in abundance, which leads to the development of the mucous rattle. The same general development and long continuance of the rhonchi is observed in the bronchitis which attends continued fever.

This mucous rattle in bronchitis, is highly characteristic when it exists. It is always double, and is always heard in its fullest development, at the base of the lungs. In cases where it is more extensive, for it may exist in every part of the lungs, it is, still, always most distinct at the base, diminishing as you recede from this point. This is one of the great diagnostic facts in auscultation. It is not perfectly explained by the examination of the bronchi after death, for other tubes, besides those in the lower lobes of the lungs, may be equally inflamed, and yet no mucous rattle have been developed in them during life. I think the de-

more marked, with the same listlessness, loss of appetite, and indisposition to play, and uneasy sleep at night, you must examine the lungs; for pneumonia or pleurisy is forming there. On examination, you may detect dulness on percussion, bronchial respiration, and other unequivocal evidences of pulmonary obstruction; and the child may die in a few days with these formidable complications. It is worthy of particular notice, that when these complications occur, either in the head or in the lungs, the whoop may very much diminish in intensity, or entirely disappear, and thus lead a careless observer to suppose that the disease was improving, while, in truth, the apparent relief is actual danger.

The examination of the chest in most cases of bronchitis, establishes the absence of physical signs rather than their existence. In all cases of simple bronchitis, however severe, there is always undiminished clearness on percussion. In some of the complicated cases, as in emphysema of the lungs, the sound on percussion may be unnaturally clear. In many cases, even when the disease assumes a suffocative character, the same clearness on percussion may be noticed; when a principal bronchus is obstructed for a time, dulness does not exist. The truth is, that, even with marked dyspnœa, there is air enough in the cells; the difficulty is, that it does not escape from them readily during the feeble act of expiration, and is not so rapidly changed as the wants of the system require. When, therefore, you find the slightest dulness on percussion, you may be certain that something else besides bronchitis is present. If it be slight, limited in extent, and situated under the clavicles, it probably depends on tuberculous deposits in the lungs. If it be more extended, it may be dependent on the existence of dilatation of the bronchi, on pulmonary œdema, on pneumonia, on pleurisy, or hydrothorax. This simple fact, the absence of dulness on percussion, is often of the greatest value, both in the diagnosis and the prognosis of pulmonary disease. A chest that sounds clearly on percussion is seldom in a very serious condition, unless, indeed, it is unnaturally resonant, as in emphysema, or in pneumothorax.

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trated by the practice of auscultation. In simple cases, you will seldom detect any decided alteration in the natural respiratory sounds. In the early stage of the disease, the respiratory murmur may be a little harsh, and an occasional sibilant or sonorous rhonchus may be detected; obstruction of the bronchi, from mucus, or from spasmodic contraction, may render the respiratory murmur temporarily feeble in the portions of lung affected by the disease: but this transient effect usually disappears after coughing, to return again, perhaps, after a short time. As the expectoration becomes more abundant, these physical signs usually disappear. In the more severe cases, a mucous rattle may be heard at the base of the lungs—*always at the base of both lungs.* It is in the complicated cases, in secondary bronchitis especially, when connected with long-continued obstruction to the pulmonary circulation, as in emphysema, and in heart disease, that you will find the best-marked physical signs of bronchitis. In these cases, during an acute attack, the rhonchi are apt to be heard abundantly all over the chest, and to continue for a long time. It is in these cases, also, that the mucous rattle is most apt to be heard, after the attack has continued for a time. The obstruction to the circulation in these cases, seems to impress a peculiar irritability on the bronchi, by which the tendency to spasmodic contraction is materially increased; hence the development of the rhonchi. The same congestion seems also to favor the secretion of mucus in abundance, which leads to the development of the mucous rattle. The same general development and long continuance of the rhonchi is observed in the bronchitis which attends continued fever.

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pendent position of the tubes in the lower lobes, must offer the true explanation. This tends to favor the accumulation of mucus in the tubes, and by favoring congestion of the tubes also, increases the tendency to secretion. That congestion favors secretion, in many cases, I think is probable, from what is noticed in the bronchi in heart disease, a fact I have already recently alluded to.

Among the rational symptoms of bronchitis, there is none more characteristic than the cough—usually sonorous, dry, and perhaps hoarse, at the commencement, but afterwards loose, with a more or less abundant expectoration. This loudness of the cough, attended by a sensation of relief, is often of great diagnostic value in the bronchitis of children, as distinguished from the short, suppressed cough of pneumonia, or of pleurisy. When, therefore, after entering the nursery, and often before this, you hear a child coughing loudly, and find, after looking at the countenance, that there is but little expression of suffering, you may feel relieved for the time at least, and on more careful examination, you will probably find nothing but bronchitis present. I would observe, however, that the cough has not always this vigorous, sonorous character. It is sometimes trifling, but then it indicates a secondary bronchitis, a most serious symptom when caused by tubercles in the lungs, a comparatively unimportant symptom when caused by gastric derangement—these being the most frequent causes of a dry, trifling cough.

The expectoration in bronchitis varies with the nature of the case. In the simple primary acute form, the expectoration is, at first, a thin, transparent, frothy mucus, often streaked by blood. This condition is soon followed by a change—the expectorated mass becoming gradually more opake, more viscid, less aerated, and without blood, until it passes into what is called purulent mucus. In certain cases of chronic bronchitis, especially if complicated by a dilatation of the bronchi, a more purulent expectoration may be noticed. In other cases, the expectorated mucus seems to be checked in its regular stages, and remains thin and transparent for a long time. This is the case when the increased secretion depends more upon secondary irritation for its cause, as in the early stage of tubercles, and in

cases of gastric derangement. Sometimes the mucus continues white, while it loses its transparency and becomes more viscid, assuming a pearl-like appearance. In emphysema, the bronchial expectoration is apt to assume a dirty look, like a solution of impure gum-arabic.

The quantity of mucus expectorated varies greatly in different cases. In most cases, both of acute and of chronic bronchitis, it is moderate in amount, seldom exceeding half a pint in the twenty-four hours. Many persons affected by chronic bronchitis, expectorate only in the morning, after rising, a small quantity of mucus. Sometimes, however, the expectoration is excessive, and this may occur both in the acute and in the chronic form of the disease. The circumstances that favor this excessive expectoration, are considerable strength in coughing, an extended bronchitis, and, more than any thing else, I think, a congestion which favors secretion.

The expectorated mucus is usually without taste or odor. Sometimes, however, it is described as being saltish, or sweetish; sometimes it is offensive, especially when it approaches pus in appearance. It might be mistaken for a gangrenous odor, but it is different from that. It is like the odor produced by an ill-conditioned ulcer.

In the early stage, it is very common to find a few streaks or spots of blood in the expectoration, not mixed with it, but lying upon it. This streaky, unmixed blood in the expectoration, distinguishes the expectoration of bronchitis from the more abundant hemorrhage of tubercles on the one hand, and the rusty expectoration of pneumonia on the other hand.

Pain in the chest is not a prominent symptom of bronchitis. During a few days after an acute attack, there is a feeling of soreness and of heat, or a sensation of weight under the sternum, but this soon subsides. When, therefore, there is pain in the chest, especially in the lateral portions, increased by a full inspiration and by coughing, you will have reason to infer the existence of some other disease, a pneumonia, or a pleurisy, unless, indeed, as may happen, the bronchitis is associated with a rheumatic affection of the muscles of the chest.

The dyspnœa is usually moderate in simple bronchitis. In

the acute capillary bronchitis of children, however, it may be intense, even suffocative. It also often possesses the same characters in the acute secondary forms of the disease, particularly in emphysema and in heart disease. In continued fever, an unusual degree of dyspnœa will usually be found to be dependent on a general bronchitis. In chronic bronchitis, this symptom is hardly experienced unless after an unusual effort, as running up stairs, or when considerable dilatation of the bronchi exists. In some of the forms of dry catarrh, where the more minute bronchi are obstructed by swelling, or by spasm, a more decided dyspnœa may exist, and become aggravated to a paroxysm of asthma, by the supervention of an acute attack. Indeed, in all the forms of chronic bronchitis, where an acute attack supervenes, the dyspnœa is usually considerably increased.

Bronchitis is chiefly to be distinguished from pneumonia and pleurisy in the acute attacks, and from tuberculous disease in the more chronic forms of the disease. The character of the cough, of the expectoration, the existence of pain, the degree of febrile excitement, are important elements of diagnosis in the first class of cases. Pneumonia and pleurisy are usually much more severe forms of disease than bronchitis. There is more febrile excitement, greater prostration, more disturbance of the digestive functions, a different kind of cough, and frequently, in pneumonia at least, a highly characteristic expectoration. The physical diagnosis is also very distinct. The absence of dulness on percussion, especially after the attack has continued a few days, is a highly important element in the diagnosis. The modifications of the respiratory sounds, the bronchial respiration in pneumonia, or the absence of the respiratory murmur in pleurisy, are very different from the vesicular murmur masked, perhaps, by a rhonchus or by a mucous rattle, which you will notice sometimes in bronchitis. But, as I have already stated, the absence of all physical signs is more frequently characteristic of bronchitis. These important, and usually easily recognized differences, can, however, be better appreciated after a careful study of the history of pneumonia and of pleurisy.

The distinction between bronchitis and tuberculous disease, can also be better understood on some future occasion. The

emaciation and loss of strength, the pain in the chest, the tendency to hemopthisis, the hectic, serve to distinguish most cases of tuberculous disease; while the physical signs, having their chief seat at the summit of the lung, are equally significant. The limited dulness, the prolonged and bronchial respiration, the detection of a cavity, the existence of a mucous rattle under the clavicle or above the spine of the scapula, can seldom mislead you in the diagnosis of this more serious disease. The latter symptom, in particular, is very diagnostic. A mucous rattle at the summit of the lungs, is usually tuberculous in its origin. A mucous rattle at the base, is usually bronchial. There are but few exceptions to this rule. It is in cases of chronic bronchitis complicated with dilatation of the bronchi, that the diagnosis is most difficult. I have already alluded to this subject. The physical signs may be quite similar, and the constitutional and rational symptoms may, also, bear a close resemblance in this affection, and in tuberculous disease. The chief points of diagnosis in these obscure cases, it will be remembered, are, the age of the patient, the diffusion of the physical signs, the absence of hemopthisis, and lastly, a point, however, of great importance, the comparative rarity of the bronchial disease.

The diagnosis of that form of secondary bronchitis dependent upon gastric derangement, from tuberculous disease, is also important. The principal points are these—the previous existence of dyspeptic symptoms, a tendency to despondency, a fear of evil consequences, an unaccelerated pulse with a tendency to cold extremities, but little emaciation, and particularly the absence of all physical signs.

The diagnosis of a dry catarrh from emphysema of the lungs is often attended with much uncertainty. The two affections are usually associated, and it is probable that either may induce the other, although it is not probable, as Laennec maintains, that the dry catarrh always precedes the emphysema. In emphysema, you will find the same rational and constitutional symptoms as in the dry catarrh; that is to say, cough with trifling or with no expectoration, permanent dyspnœa, aggravated to a paroxysm of asthma by an acute attack of bronchitis, feeble respiration, and sibilant rhonchus. But in the former disease, the feeble

respiratory murmur is constant, or a harsh respiration exists; there is also unusual clearness on percussion, and often dilatation of the parietes of the chest. I believe also that in emphysema, it is usually the anterior and superior portions of the chest that exhibit the physical signs most distinctly.

The *treatment* of acute bronchitis, even when sufficiently severe to call for the interference of the medical practitioner, is very simple. To moderate the febrile excitement by saline purgatives and by antimonials, to act upon the skin by diaphoretics, to keep the patient quiet in bed, and to recommend a low diet—these are the chief indications. Bleeding, in any form, is only necessary in cases in which the disease assumes a suffocative character, or in which there is an evident tendency to pneumonia or pleurisy. It is very seldom advisable to take blood from the arm, unless in cases of heart disease, where obstruction to the circulation is a principal difficulty. Leeching, or cupping, is much more frequently indicated. In the capillary bronchitis of children, in the milder form of suffocative catarrh from obstructed circulation, in threatened pneumonia or pleurisy, immediate and great relief often follows the local abstraction of blood. If, however, the pulse is full and strong, the fever considerable, and, above all, the patient plethoric, there may be a decided advantage in first opening a vein in the arm.

Much advantage in the early stage will be found in the free and repeated use of the tartar-emetic, in doses sufficient to create a moderate degree of nausea. If there be evidence of a considerable accumulation of mucus in the bronchi, much relief will be experienced by occasionally vomiting the patient. The free expectoration of mucus, which attends this effort, explains the relief obtained. You will remember that in the early stage of bronchitis, there is a tendency to spasm of the bronchi, as indicated by the rhonchi present, and the relief obtained by nauseating doses of tartar-emetic will be proportioned to the abundance of these rhonchi. The influence of this remedy, also, in causing relief by subduing the febrile excitement, must be apparent to all those familiar with its use. In many cases, a still milder treatment is indicated. I am in the habit of combining ipecac with the Spirit of Mindererus with excellent ef-

fect in mild cases of acute bronchitis. Indeed, it frequently happens, when the bowels are irritable, or the patient very young and feeble, that antimony is contra-indicated.

As a general rule, you should abstain from the use of opium in the early stage of bronchitis, as it seems to check the tendency to secretion, which it is your object to promote, and which is generally followed by decided relief. Combined with ipecac or antimony, it is undoubtedly less injurious than when given alone. It may, sometimes, be decidedly beneficial, particularly in nervous, irritable subjects, in whom restlessness and distress are disproportioned to the other symptoms. A Dover's powder at night, in such cases, will induce sleep, and add much to the comfort of the patient.

Counter-irritation to the chest is sometimes very beneficial, especially where the bronchitis assumes a suffocative character, and the febrile excitement is not considerable. Indeed, in many of these cases, the heat of the skin is very moderate, and the extremities may even feel cool, and the pulse be feeble. In these cases, nothing can be better than the free use of mustard to the chest, as well as to other parts of the body, or the warm bath, rendered stimulating by mustard.

The warm bath is a valuable remedy in acute bronchitis, if used with care. In the cases of children especially, the greatest care is necessary that they are not chilled in being removed from the bath. The following precautions are of importance: see that the temperature of the room is at least at eighty degrees, and that of the bath at one hundred degrees; place a blanket around the neck, and over the bathing-tub, while the patient is in the bath. Receive the child, on leaving the bath, in a well-warmed blanket, and place it in bed; do not stop to dress it, or even to wipe it. Generally the child will fall asleep, and a gentle perspiration will frequently indicate the good effect of the treatment.

After the acute symptoms of bronchitis are relieved, the stimulating expectorants may be employed with advantage. This class of remedies is large, and they are frequently used in combination. The squill, senega, sanguinaria, tolu, are those most frequently used. It is common, also, to combine them

with a proportion of opium. A still more powerful class of expectorants exists in the gum ammoniac and the carbonate of ammonia. The latter especially, may be used with great advantage in the bronchitis which attends typhoid fever, and whenever the vital powers are much reduced, as by old age, or by other debilitating causes.

Blisters to the chest generally do no good in acute bronchitis, if applied early in the disease; but it is not unusual for a protracted case to yield readily to a well-drawn blister, when other means have failed. A stimulating wash to the chest, into which turpentine enters largely, is often useful in these cases.

Chronic bronchitis is not, in my opinion, generally much under the influence of remedies. The variable condition of our climate is such, that when the disease is long established, the bronchial membranes become so irritable, that a trifling exposure will aggravate the case, and thus prolong it indefinitely. There are many individuals, who nearly cease to cough during the summer season, but who relapse again as the winter advances. The best remedy is a sea-voyage, and a protracted residence in a mild and equable climate—a dry atmosphere, if the expectoration be profuse, in a more moist atmosphere, if the cough be dry, should be preferred. If the disease is secondary, especially if connected with emphysema, much may be gained by this change, but it must be permanent—a fresh exposure will lead to a relapse. The form of chronic bronchitis most amenable to treatment is that dependent upon gastric derangement. A removal of the gastric difficulty carries with it the bronchitis. It is in these cases, especially, that a voyage effects so much, such permanent good.

The stimulating expectorants are much used in chronic bronchitis—the same class of remedies that I have briefly mentioned as useful in the advanced stage of the acute form. Other remedies have, also, been much used; as the balsam of copaiba, the benzoic acid. But these remedies are disagreeable to most patients, and are seldom long continued. It was formerly the practice to employ repeated emetics in this disease; I believe, if successful, that they operate principally in removing a gastric derangement on which the bronchitis depends. Laennec thought

very highly of the repeated use of emetics in chronic bronchitis, as calculated to relieve the lungs of an accumulation of mucus, and to impress a new and favorable action on the mucous membrane. He also advocated the use of alkalies in the dry catarrh, as calculated to render the secretion less viscid. Most cases are, I think, best managed by occasional palliatives when the symptoms are troublesome, and, above all, by a careful regimen. The greatest care should be taken to guard against sudden exposure, and a damp night-air. The chest should be fortified by daily ablutions, cold or warm, as the case may be, and by the free use of friction to the chest. The diet should be simple, and such as is unstimulating and easy of digestion. Jeffries' respirator is, I think, an excellent contrivance for irritable lungs. It need not be worn constantly, but may be carried in the pocket, and used whenever the air is found to be too irritating.

There are some cases of chronic bronchitis which, I think, are much benefited by the use of tonics, especially by the preparations of iron. I have used them with good effect in delicate subjects, in whom a predisposition to phthisis is suspected. The iodide of iron is, perhaps, the best form to employ in such cases. Care should be taken to begin with small doses, as it sometimes, if too freely employed, acts as an irritant, and increases the cough.

The hydriodate of potass, in large doses, has been highly recommended as a remedy for the bronchitis which accompanies emphysema of the lungs. If it relieve at all, it must do so by improving the accompanying bronchitis. I suspect that the cases in which it has been found most productive of benefit are cases of the dry catarrh of Laennec, an affection often associated with emphysema.

The treatment of whooping-cough is, generally, extremely simple. As in most diseases whose duration is self-limited, the expectant treatment is the most proper. The patient should be guarded against undue exposure, especially by warm clothing; the diet should be restricted and simple, and then the case should be watched. There is no reason why children, if the weather is fine, should not go out into the open air. They are probably the better for it, if they are carefully watched and

properly attended. Certain cases, however, are benefited by medical treatment. If much ordinary bronchitis is present as a complication, this should be relieved by the ordinary means, and care be taken that it does not pass into pneumonia. If the paroxysms are unusually severe, their violence may be mitigated by sedatives, as belladonna, and other remedies of the same class. Much has been written on the influence of the belladonna in abbreviating the duration of the disease. I doubt very much if this is true, but it may serve as a useful palliative. If the symptoms of congestion of the brain ensue, or those of pneumonia, they should be met by the most prompt and efficient remedies adapted to these conditions; and parents should always be instructed to watch for their earliest appearance, and to take the alarm at once.

Sometimes cases of whooping-cough assume a chronic form, continuing, after several relapses, perhaps much beyond the usual period. In these cases, tonics are indicated. The mineral acids, the preparations of iron, quinine, may be used with advantage, also stimulating frictions to the spine. But the great remedy is a change of air. Few cases resist the speedy influence of this change.

LECTURE V.

PNEUMONIA.

Its different stages.—Congestion.—Hepatization.—Suppuration.—Abscess of the lung.— Gangrene.— Splenization.— Carnification.— Chronic pneumonia.— Varieties in the seat and in the extent of the disease.

Pneumonia is, primarily, an inflammation of the air-vesicles, although in all cases the surrounding tissues of the lungs are affected, and usually at the very onset of the disease. The inflammation extends rapidly to the intervesicular cellular tissue, to the bronchi, and to the pleura, so that pneumonia may be regarded, in one sense, as an affection of the whole lung.

The disease presents, in its ordinary form, three distinct stages. The first stage is that of inflammatory *congestion*, in which the capillaries of the lung become loaded with red blood, and the tissues become slightly softened and infiltrated with a reddish and rather turbid serum, holding fibrine in solution. If you examine a lung thus affected, you will find it of a reddish-brown color, somewhat swollen and softened, crepitating less than natural, but still able to float on the surface of water; pitting on pressure, and giving out from its cut surface an abundant, reddish, turbid, frothy serum, which escapes chiefly from the air-cells. The lungs of almost all who die, especially after a long agony, present these appearances in their postero-inferior portions; so that this condition has been regarded, in many instances, as the effect of simple stasis of the blood from position, aided by weakness, and by obstruction to the pulmonary circulation—an affair of the agony, and, perhaps, even occurring in some degree after death. Attempts have been made to point out the diagnostic differences between a simple stasis of the blood and inflammatory congestion. I have myself endeavored to detect some differences, but after the most careful examination I have been compelled to acknowledge the appearances as identical. Some have said, that in the inflammatory congestion the tissues were softened, while in congestion from simple stasis they were not; but this is not the case. The microscope may aid you. (*Note to Pleurisy.*) Cruveilhier, indeed, has explained the difficulty, by maintaining that the two conditions are identical in nature as well as in appearance—that they are, in fact, both owing to inflammatory congestion—the one, occurring during the last moments of life, and usually limited to the most dependent portion of the lungs; the other, occurring at an earlier period, and affecting every portion of the pulmonary substance, and almost always associated with the second stage of the disease.

Pneumonia, indeed, passes rapidly into the second stage; so rapidly, in fact, that it is rare not to find it in cases which have exhibited any symptoms of the disease during life. The lung retains its dark-red color, or perhaps, becomes of a brighter hue. It is swollen, heavy, uncrepitating, and feels solid like

the liver, when pressed by the fingers. The color, and the compact solid feeling of the lung, has given to this stage of the disease the name of *hepatization*. When the substance of the lung is divided by the scalpel it cuts like a solid, but is divided with ease, and the surface appears smooth and rather dry, and colored by different shades of red, some being darker than the rest. On pressure, you will discover a moderate exudation from the divided surface, of a slightly viscid, reddish, unaerated fluid, and in attempting to tear the lung you will find its tissues softened. If an affected portion be thrown into water, it sinks at once to the bottom. If, again, you examine the cut surface carefully, you will find it granulated; or if this is not evident, it can, generally, be made so by tearing the lung, when the surface will be found studded with small, red, rounded granulations, which are, in fact, the air-vesicles filled by a reddish lymph. In some cases, this granular appearance is not distinct, owing to the fact that the granulations are so closely packed, either by the distension of the cells, or by a swelling of the pulmonary tissues, that they appear to coalesce. According to the observations of Valleix, these granulations do not appear in the pneumonia of new-born children, the cut surface being smooth, shining, and even polished like marble. The portion affected is indurated in most cases; occasionally, it is found softened and friable, as it is in adults.

In the third stage of the disease, the color of the lung changes. The cut surface, still solid, is marbled red and gray, and finally, gray and yellow. If pressed, its tissues are evidently still more softened than in the second stage, and a fluid, evidently purulent, exudes on pressure. The lung is destitute of air, and sinks readily in water, as in the second stage. Examined carefully, it still presents the granular appearance, but the granulations have become more or less yellow. In a well-developed condition of this third, or *suppurative stage*, the lung assumes a straw color, and its tissues are so much softened, that they break down on the slightest pressure into a purulent detritus. Indeed, a complete disorganization has taken place. Yet, singularly enough, with this condition of things a well-formed abscess of the lung is of very rare occurrence. Ab-

scesses have, however, been found in all parts of the lungs, usually, of considerable size, irregular in shape, as if formed by the breaking down of the surrounding parts towards a common centre, sometimes traversed by bands containing obliterated vessels, or tubes. Sometimes the abscess is invested by a false membrane, at other times surrounded directly by a mass of purulent infiltration. Usually, the abscess is single, but sometimes multiple, especially in children, when this result is the consequence of a lobular pneumonia. I have never yet met with an instance of abscess as the consequence of simple pneumonia; it is very rare. There is reason, however, to think that it is sometimes produced, artificially, in a lung in the suppurative stage, by external pressure with the fingers in removing the lung where it adheres to the ribs. Indeed, nothing is easier than to produce an artificial abscess in a suppurating lung, by pushing the finger into its substance, thus forming a cavity, which is at once filled by pus. These abscesses sometimes become perfectly cicatrized. Dr. Stokes has given an example in his work, of an individual who died of a second attack of pneumonia a year after the abscess had formed. The lung where the abscess had existed was united to the ribs by old adhesions. Its summit was much puckered, and a cartilaginous mass, three inches in length and half an inch thick, existed in the neighborhood of the puckering, and which consisted of two layers loosely united by a fine cellular tissue.*

Inflammation, in certain rare cases, attacks the cellular tissue of the lung and passes into diffused suppuration. In this case, the pleura may be dissected from the lung, the lobules from each other, and even the pulmonary vesicles have been drawn out from the suppurating mass, hanging to the bronchi like grapes to their stem. It has been proposed to call this rare form of inflammation the *dissecting abscess.*

There are two circumstances, however, which seem to favor the formation of abscesses in the lung. The first is an effusion

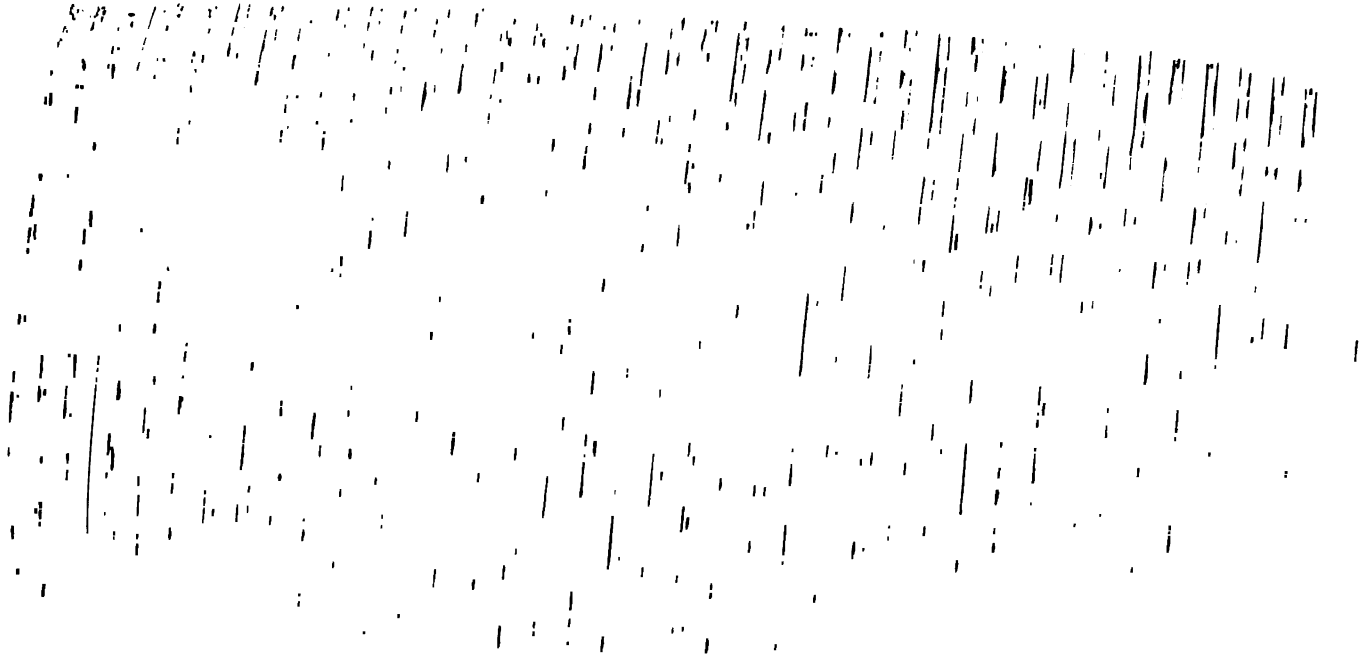
* For a condensed account of the successive changes which effused lymph undergoes—its absorption, its conversion into pus, and its transformations as an organized tissue, as observed under the microscope, the student may consult the note to the pathological anatomy of pleurisy.

of pus into the cavity of the pleura. Where this has occurred, it will sometimes happen that a distinct abscess will form in the lung, which may, finally, open into a bronchus, or communicate with the pleura. In the second case, the lung may be studded with small abscesses. These abscesses are peculiar, both in their cause and in their mode of formation. They are called *metastatic* abscesses, and caused by the purulent infection of the blood. According to Cruveilhier, the pus globules are transported by the circulation from their remote source to the capillaries of the lungs, some of them are detained there, and each arrested globule excites a capillary phlebitis with a local pulmonary apoplexy, which leads to the formation of a firm nodule, varying from the size of a mustard-seed to that of a hazelnut, of a blackish color, usually situated superficially, and in the lower more frequently than in the upper lobes. These dark nodules soften in their centre, a deposit of pus takes place there, and a small circumscribed abscess is formed, sometimes invested by a false membrane, at other times not. Sometimes, it is surrounded by healthy or slightly congested lung; at other times, by lung in a state of hepatization.

According to Rokitanski, these nodules may undergo another change in very rare cases. They may be converted into a grayish, hard nodule, which becomes inclosed in a cellulo-fibrous capsule, and which may, in time, be converted into an osseous concretion.

The three stages of inflammation are frequently found united in the same lung, but the second stage is usually predominant. Frequently nearly a whole lung is found hepatized, with a limited portion in the first stage, or passing into the third stage. In most fatal cases, a tendency to the third stage is exhibited in the gray and red marbling, and the increased softening of the pulmonary tissues; but a case in which a well-marked suppurating lung is found to be the predominant lesion, is, I think, comparatively rare, not because the tendency to this condition does not exist, but because death, usually, occurs before this stage is fully developed.

Inflammation, when it attacks a lung, tends to spread in every direction, although, perhaps, rather more readily over the



original form of the disease can easily be detected. The lung still presents a more or less uneven surface, and its nodulated feeling is not entirely gone.

Finally, there are cases, very rare indeed, in which the inflammation seems to be limited to individual vesicles, and single granulations precisely like those found compacted together in ordinary inflammation, are scattered through the pulmonary tissues, surrounded by lung perhaps a little congested. There is no satisfactory reason known for this rare form of the disease.

I have thus explained to you, that pneumonia, when it exists in its ordinary form, presents certain conditions which belong to a continuous inflammation, affecting, almost simultaneously, all the tissues of the lung, while, in certain cases, it assumes a lobulated appearance, from the fact that separate lobules are affected, and at different periods of the disease. I have also stated that the chief cause of this difference seems to be, that in the latter case, the disease is first a bronchitis of the capillary tubes, and that the inflammation spreads from these tubes to the air-vesicles supplied by them. Each lobule, in fact, is a little lung, and the different parts composing it are in intimate relation to each other. Each has its own particular bronchus, and the air-cells of each, perhaps, communicate with each other. So that you can readily understand why an inflammation, originating in a particular lobule, should, after rapidly affecting that lobule, be at least partially limited in its extent by the surrounding cellular tissue. It is important to remember that this lobular inflammation occurs, chiefly, in children from one to six years of age.

But there are other causes which modify the appearances of an inflamed lung. The condition of the blood itself, and the general state of the solids, modify the condition of the lung. Thus, in typhus fever, the softening of the tissues is more marked, and the color of the lung is darker, and the same is true in other cases, where the typhoid condition exists. While, if the disease is slowly developed, especially in the lobular pneumonia of debilitated children, the lung is unusually dry and the tint bluish. In intemperate persons, the tendency to the third, or suppurative stage is more marked, while, in secondary pneumonia, the reverse of this is true.

There are certain conditions of the lung in which the appearances differ so much from the usual consequences of inflammation that they have excited particular attention, and have received distinct names. One of the most striking, is that known as *splenization* of the lung, a condition not very infrequent in continued fever. In this case, the portion affected, and it is usually the base of the lung, is swollen, bluish, or dark red. It is very much softened, giving out, when pressed, a viscid, grumous, dark red, unaerated fluid. The lung, in fact, resembles quite as much the spleen, as it does the liver in cases of hepatization. This condition is pneumonia in the first stage, or more properly the imperfectly developed second stage, united with venous congestion and unusual softening of the tissues.

Another condition, not unfrequently noticed, is that in which the lung presents an appearance not unlike muscle, whence it has been called *carnification*. The lung is red, flaccid, unaerated, is torn with difficulty, and when its cut surface is pressed nothing but a little blood exudes. This condition occurs in a lung which has been first compressed by an effusion into the pleural sac, and in which a slight capillary congestion has ensued.

It is usual to class *gangrene of the lungs* among the consequences of inflammation. It is doubtful, however, whether this is correct, at least in a majority of cases. It seems to me that gangrene of the lung resembles very much the gangrene of the toes in old people, and I have long suspected that it originates in the same cause, viz., a loss of nutrition from obliteration of the arteries. The bronchial arteries which nourish the lung are remarkably small and long vessels, and well suited to become the seat of obstruction from a variety of causes. Yet no one has as yet established the fact whether they are obstructed or not in gangrene of the lung. The subject is worth a careful study.

The most common form of gangrene appears as a circumscribed, blackish, or greenish-black eschar, as if produced by some strong caustic. It is, usually, quite limited, although by no means uniform in size. It tends to break down into a greenish and horribly fetid deliquum, containing ash-colored portions

of dead cellular tissue. This process begins at the circumference, so that you may find in the centre of the decomposing mass a blackish spongy substance—the dead lung in a less advanced stage of dissolution. The walls of the cavity may be formed by bare pulmonary tissue hepatized, infiltrated by the decomposing fluids, or they may be formed by an organized false membrane, which, from its firmness, thickness, and perfect organization in some cases, must have existed for a considerable time.* Usually, the inflammation extends far beyond the immediate neighborhood of the gangrenous mass: it may affect a whole lobe, or a whole lung.

The gangrenous abscess thus formed usually opens into a bronchus, and the contained matter is then expectorated; or it may open into the cavity of the pleura, and pneumothorax follow; or a large pulmonary vessel may be corroded by the gangrene, and fatal hemorrhage ensue. Either from the difficulty in expectorating all the gangrenous mass, or from the extent of the surrounding inflammation, or, from the supervention of pneumothorax, or of hemorrhage, you will, usually, find the work of reparation hardly, if at all, commenced. The gangrenous mass, partially softened, partially evacuated, will be found with its horrible odor, and the surrounding lung in the second stage of inflammation, which has spread far from the gangrenous portion in the neighborhood of which it had originated. In certain rare cases, the work of reparation may be complete.

* Many years ago, I removed the lung of a patient, who had died with the symptoms of gangrene of the lungs, which had existed only for a few days. A large mass of diffluent gangrene was found in one of the lungs, invested by a thick, well-organized false membrane, which must have required a considerable time for its formation. The surrounding lung was inflamed. Since that time I have met with a case of gangrene of the liver, also inclosed in a firm, well-organized sac, at least two lines in thickness. In this remarkable case, the gangrenous eschar, composed of dead cellular tissue, and of small obliterated blood-vessels, was entirely free from any gangrenous odor. The surrounding liver was healthy, except that the cellular tissue about the vessels of the liver was much thickened and condensed, and *only* in the immediate neighborhood of the gangrenous mass. It seems to me not improbable, that this was the original condition, only to a greater degree, at the seat of the gangrene, and that the compression of the hepatic arteries from this cause may have been the cause of the gangrene. The affection was entirely latent, the patient dying of empyema.

The gangrenous mass is invested by a false membrane, is evacuated, healthy pus is secreted, the surrounding inflammation subsides, contraction and cicatrization ensue.

Laennec has described a form of gangrene of the lung, which he calls the uncircumscribed gangrene. He had met with but two cases. For myself, I have never seen one case. According to Laennec's very graphic description, the gangrenous portions are irregularly diffused, passing, insensibly, into portions which are congested, as in the first stage of pneumonia. In this condition, the lung presents the differing shades of a dirty white, or greenish, or blackish color, mixed with portions of a livid red, while the whole of the lung affected is more moist and friable than natural, and exhales a decided gangrenous odor.

Gangrene may, also, attack the walls of the simple pulmonary abscess, or of the tuberculous abscess, or the mucous membrane of the bronchi, which then becomes soft, and of a brownish-green color, either in spots, or over a diffused surface.

Softening of the lung is sometimes met with, which resembles very much gangrene in appearance, but wants entirely the characteristic odor. The part affected is soft and moist, breaking down by slight pressure, into a pulp of a dirty brown, or blackish, or grayish color.

Chronic pneumonia is a very rare form of disease. In certain cases, however, a portion of lung, sometimes a lobe, or even a larger portion, passes into this condition from the stage of hepatization. The red color gradually changes to a grayish, slaty, or even blackish tint, from the admixture of black pulmonary matter, the granulations disappear, induration ensues, and the interlobular cellular tissue becomes more distinct and fibrous in its character. The lung in this condition is firm and solid, and intersected by white lines, from the development of the cellular tissue, and from the obliteration of the bronchi. The same appearances are not unfrequently noticed in the tissue surrounding tuberculous abscesses, but limited in extent. In certain cases, which I will now allude to, the development of the cellular tissue is the principal step in the series of morbid changes; but then, the disease is not to be regarded as the consequence of pneumonia. Inflammation primarily attacks this tissue, which

may pass into suppuration, constituting the dissecting abscess of which I have already spoken. In other cases, however, the inflammation is more chronic in its character. There is a deposit of lymph in the cells of this tissue, which, becoming organized, gradually causes a shrinking of the tissue that obliterates the air-cells, the smaller bronchi, and blood-vessels, and converts the lung into the cellulo-fibrous mass called cirrhosis, and of which I have already spoken in a former lecture, as associated with dilatation of the bronchi.

Pneumonia, when primitive, is commonly confined to one lung: in a few cases it attacks both lungs, but not at the same moment of time. The pneumonia which follows bronchitis is, on the contrary, commonly double, as in the form most frequently observed in young children. The same tendency to double pneumonia exists in new-born children, and without being preceded by bronchitis. The right lung is more frequently attacked than the left lung, and the proportion is too considerable not to be worthy of remark. The disease most frequently commences at the base of the lung, and affects the posterior rather than the anterior portion. It is not uncommon, however, for the disease to attack the middle portion of the surface of the lung, and occasionally, but this is rare, it attacks the central portion of the organ.*

* The French writers have applied the valuable aid of medical statistics to elucidate the seat of pneumonia. I derive the following statistics from Grisolle. In 280 cases, observed by himself, the disease was confined to the right lung in 166; in 97 to the left lung; it was double in 17. The cases occurred among adults. Rilliet and Barthez, whose observations were confined to children from the age of one year to 15 years, prove that lobular pneumonia is almost always double by the following statistics. Thus, of 203 patients, in whom this form of the disease existed, in only five was the disease confined to one lung. In the lobar form of pneumonia, however, occurring chiefly among the older children, and resembling the most common form of the disease in adults, we find the rule observed in adults confirmed. According to these authors, of 84 cases, the disease was in the right lung in 48; in the left lung in 27; and in both lungs in only 9 cases. In the 75 cases of single pneumonia, the disease occurred 48 times at the base and 27 times at the summit of the lung. These authors give us no statistics of the relative frequency of the lobular pneumonia in the upper and lower lobes, but express the opinion that it is decidedly most frequent in the lower lobes. The same general facts have been noticed in the pneumonia of new-born children, as observed by Valleix and by Vernois. Thus, in

Bronchitis is an almost constant *complication* of pneumonia, at least in the adult; and Rilliet and Barthez have deserved great credit for establishing the fact of its very frequent occurrence, even as the precursor of pneumonia in children. The same fact—bronchitis extending to the air-vesicles, and inducing pneumonia—is by no means rare in adult life, a fact which should teach you never to neglect a bronchitis; as a secondary complication, however, it is almost always present. It is true that the inflammation of the bronchial mucous membrane may not be very strongly marked, although I have usually found it so in the neighborhood of the portion of lung affected by pneumonia. But you must not be guided entirely by the anatomical appearances. Certain it is, that a considerable expectoration of bronchial mucus, more or less opake, occurs in almost every case of pneumonia, in which the expectoration is noticed, and an unusual secretion of the same is found in the bronchi after death. I mention these facts the more particularly, because some excellent observers of recent date have been disposed to underrate the existence of bronchitis in this disease. I refer particularly to Grisolle. Valleix, also, considers it rare and of trifling importance in the pneumonia of new-born children.

You will very commonly find the bronchial glands at the root of the lung inflamed—that is, red and softened.

Another complication which rarely fails to exist, at least in the pneumonia of adults, is pleurisy. In certain rare cases of central pneumonia it may not be found. But when the disease affects the surface of the lung, as it usually does, pleurisy is sure to follow; and in most cases, it evidently follows closely the

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128 examinations, the disease was double in 111; and where it was single, it was always seated in the right lung.—In 31 cases, 13 were *under* 45 years of age; of these, 11 had pneumonia of the inferior lobe, and 2 only, pneumonia of the superior lobe. In 18 cases *above* the age of 45 years, 8 had pneumonia of the inferior lobe; 8 pneumonia of the superior lobe; 2 pneumonia of the middle lobe. The same tendency to pneumonia of the upper lobe would seem to exist at the other extreme of life, if we examine the statistics of Valleix. Thus, in 139 cases of pneumonia in new-born children, the superior lobe was affected alone in 20 cases, while the inferior lobe was affected alone in 44 cases; both lobes were affected in 44 cases. In the remainder it was more or less spread through the lung

original disease. It possesses some peculiar characteristics. It is usually a dry pleurisy; that is to say, it causes a simple exudation of lymph, and often in very small quantity. Indeed, so thin and transparent is the coating of lymph, in many cases, upon the inflamed lung, that it might easily escape detection. The substance of the lung beneath, appears nearly as distinctly as is natural; but with the scalpel you can readily raise a thin pellicle of recent lymph from the inflamed surface. Frequently, indeed generally, the effusion of lymph is more abundant, and both opake and yellowish, forming an irregular coating over the affected portion. It is rare to find a considerable effusion of serum, as happens in simple pleurisy. Sometimes, however, a moderate quantity is noticed. It would appear from observation, that this secondary pleurisy is comparatively rare at the two extremes of life, in young children, and in those of an advanced age.

The heart is usually found more or less congested by blood in fatal cases, especially the right cavities of the organ, and even clots of fibrine, free from the red portions of the blood, are not uncommon. I have, frequently, also noticed that the larger pulmonary vessels in the lung were more or less obstructed by coagula of fibrine, which could easily be pulled out from the vessels by the forceps. It is probable that the inflammatory nature of the blood, abounding in fibrine, the stasis of the blood in the vessels, and perhaps an extension of a low degree of inflammatory action to the lining membrane of the vessels, are the causes of the frequent formation of these coagula.

These are the chief organic complications noticed in pneumonia. Of course, a disease producing so marked an interruption to the circulation may induce secondary congestions. The brain, especially in children, may present evidences of this condition, and the same is true of the liver and of other abdominal organs. The brain, indeed, will sometimes become affected with arachnitis, which is a most serious complication.

LECTURE VI.

PNEUMONIA.

Causes.—Influence of climate and of the seasons.—Exposure to cold as an exciting cause.—Prevalence of the disease in different professions.—Causes of secondary pneumonia.—Duration of pneumonia.—Mortality.

The causes which operate in the production of pneumonia are involved in much obscurity. Every careful observer must have frequently witnessed cases in which no obvious cause of the disease could be detected. There are, no doubt, individuals in whom there is a constitutional tendency to the disease, since you will find it occurring repeatedly in the same subject, while others, much more exposed apparently, escape it entirely during a long life. There is no evidence, however, that I am acquainted with, which tends to prove that there is an hereditary influence in the production of pneumonia. A disease so common and so easily recognized, could hardly have failed to have been branded with this influence did it exist.

Observation has proved that the disease is of frequent occurrence at every period of life, and that it prevails in every quarter of the globe. It has been noticed in the *fœtus in utero*, and its frequency in new-born children, in young children between the ages of one year and six years, and again in old age has often been remarked. Laennec observes, that the two extremes of life are most subject to its influence; they are certainly most exposed to its mortality. The adult age seems to be that in which its frequency is least observed, and especially that in which its mortality is the least marked. Again, those regions which are called temperate, but in which sudden changes of the weather are apt to occur, and, especially, those in which damp and cold winds are prevalent, are the favorite seats of this disease. Still, there is no region, however equatorial, or steady in its climate, where the disease may not be found. So there is no season of the year which is exempt from its ravages; while those seasons during which cold and damp winds prevail, and

during which sudden changes of temperature are more marked, are those in which the disease prevails. The experience of this Hospital proves that the latter part of winter, and the spring, is the season of pneumonia. Cases begin to be more numerous during the month of February, and continue to be so until June. The tendency to pneumonia is equally observed in the secondary form which accompanies continued fever. This complication is much more common during the spring season than at any other portion of the year. It has also been observed, that pneumonia when epidemic, as it sometimes is, commonly commences at the same season. These facts will enable you to establish a decided influence in the prevailing character of the climate, and of a particular season of the year; and this character is a tendency to the prevalence of cold, damp, changeable weather.

But in addition to this general influence of the seasons, its particular influence in individual cases is worthy of remark. Many persons can directly trace the attack to exposure to cold and dampness, especially when heated; and this has been regarded as the chief cause of the disease. It is, probably, the best recognized cause of the primitive form, but still there are many, and probably the larger number, who contract the disease while in apparent good health, and who cannot recognize this or any other evident cause of the attack. We owe to Grisolle much valuable information on this subject. This observer found only 49, among 205 patients affected with pneumonia, in whom the direct exposure to cold could be regarded as the efficient cause of the disease. He cautions us particularly against a, no doubt, frequent source of error. Many patients mistake the chill, which is in fact the onset of the disease, for an exposure to cold, and thus the patient, and the practitioner also, adopting the popular idea as to the influence of cold, may easily be led into error. It is, no doubt, very common for persons while sitting by their firesides to perceive the incipient chilliness of the disease, and to imagine that they must have taken cold somewhere, yet, after careful questioning, there is not the least evidence of any such exposure. That direct exposure to the influence of unfavorable weather is not necessary to the production of the disease, is proved by its frequent occurrence, at favorable seasons, in public

institutions, and even among those confined to their beds, as with continued fever.

If, however, exposure to bad weather is a frequent cause of the disease, as all must, I think, admit, it would seem reasonable that those occupations in life in which this kind of exposure is most common would be frequently marked by the disease. This is, no doubt, true in a general sense, but not in every view of the question. It would seem, that those who are exposed to a sudden change of temperature, provided the change is not of long duration, suffer less than those who, when heated, are exposed to the action of cold and moisture for a longer period of time. Thus those mechanics, who, like the mason, are exposed to the long-continued action of a cold draught of air, suffer more frequently than the baker, who is compelled to pass from his oven to a colder region, but is not compelled to remain exposed for any considerable length of time. Indeed, I am well convinced, from observation, that a draught of cold air blowing upon the person, even if it is within doors, is far more apt to induce inflammatory action than a much greater exposure to the cold in the open air. It is much more safe to walk out in a stormy day, exposed to the wind and to the rain, than to sit quietly in a warm room near a crack in the window. Many attacks are, I believe, induced by this unheeded exposure. An influence so slight in appearance, yet so serious to those disposed to inflammation, should never be overlooked, although it cannot always be proved to have existed.

The life of a sailor, although exposed to sudden changes of temperature as well as to rainy weather, does not seem favorable to the production of pneumonia. It is a common remark, that persons whose chests are delicate can expose themselves to the night air and to rainy weather with far greater impunity at sea than on shore. There is, undoubtedly, some influence in the sea which is favorable to pulmonary diseases in general. But this influence is confined to the open ocean—for when a ship reaches soundings this favorable influence ceases. It is not uncommon for sailors to be affected with pneumonia on approaching the land in bad weather, and the same unfavorable influence is noticed by invalids with pulmonary affections. It

would appear, from an extensive observation, that pneumonia is of more frequent occurrence among males than among females, the result, probably, of greater exposure in the former sex; for where the sexes are equally exposed to the vicissitudes of the weather, the difference is not striking.

Primitive pneumonia is a very common disease, but secondary pneumonia is still more so. The number of those who die both with acute and with chronic disease, and exhibit after death traces of inflammation of the lungs, is very large. In acute diseases, a chief cause of the existence of pneumonia is a high degree of febrile reaction, it being a well-established fact, that secondary inflammations are apt to occur in proportion to the degree of fever present. But, in addition to this general law, there are special causes which determine the secondary inflammation to the lungs, and among these are, as I have already stated, the unfavorable season of the year, the spring with its vicissitudes of weather, acting in a general way or after evident exposure. Again, there are certain febrile diseases in which there is a particular tendency to the lungs from some specific cause, as is remarkably the case in measles, and to a certain extent, in small-pox. Again, there are other diseases of an active character, which, having their seat in the air-passages, are more easily propagated to the air-cells. This is the case in croup, in whooping-cough, and, indeed, in all the forms of acute bronchitis. The frequent extension of inflammation from the bronchi to the air-cells is well established. In children, this has been particularly noticed. Rilliet and Barthez maintain, that even in the primitive pneumonia of young children, as it is generally called, the symptoms of bronchitis usually precede the inflammation of the air-cells; and in adults, I have repeatedly noticed an ordinary bronchitis, which has been neglected, pass, after a week, or after a longer time, into pneumonia.

But those suffering from chronic diseases are quite as liable as those affected with acute diseases to a secondary pneumonia. How common is it in the post-mortem examinations of those who have died of the most chronic affections, and in whom the vitality has, for a long time, been reduced to the lowest ebb, dying, too, with diseases affecting organs remote from the lungs,

as, for instance, with chronic dysentery, having been exposed to no sudden changes of the weather, confined, even, for a long time to bed, and yet presenting traces of pneumonia of recent occurrence. The history of these cases might well lead you to the belief that exhaustion was a chief cause of pulmonary inflammation. It certainly does not seem to be any bar to its existence. But there are other influences in chronic diseases which must not be overlooked, and which are favorable to the production of pneumonia. There is the increased susceptibility to sudden changes in the weather, the effect of debility and of confinement. But what is more important than this, is the effect of the recumbent position, which, continued for a long time, causes congestion of the pulmonary tissues, and which, as I have stated in a former lecture, cannot be anatomically distinguished from the first stage of pneumonia. Indeed, it is, by some pathologists, thought to be identical with it. This tendency to congestion lays the foundation of pneumonia. This is, perhaps, the reason why those suffering from advanced heart disease are so subject to pneumonia, viz., the tendency of the disease to produce congestion of the lungs in the first instance. A most striking fact, illustrating the influence of position upon the production of pneumonia, occurred at the Maternity Hospital of Paris, where the disease existed as an epidemic. It was the custom of the nurses to place the children on the right side, and to carry them on the left arm. It was observed that nearly all the attacks of pneumonia were in the right lung. Care being taken to reverse the position of the children, the frequency of the disease did not diminish, but its seat was changed to the left lung.

I have already alluded to the fact, that certain morbid poisons, by acting directly on the lungs, are efficient causes of pneumonia. As a general rule, their first influence is upon the bronchial mucous membrane, as in whooping-cough and measles, inducing, first, a congestion of the lungs, and then pneumonia. In other cases, a different effect is noticed, as in the case of purulent absorption, especially after amputations. In these cases, according to the experiments of Cruveilhier, pus is admitted into the veins, carried into the circulation, and the globules being

arrested in the capillary veins of the lungs, become the seat of a circumscribed obstruction and irritation, which is the starting point of the small metastatic abscesses found in these organs. The obstructed pus globule has never, to my knowledge, been detected, but the beautiful experiment of Cruveilhier throws much light on the subject. After injecting mercury into the veins of animals, he found the same metastatic abscesses, and in the centre of each a globule of the mercury. The observations of Dr. Budd, also, tend to confirm this view of the subject. According to this observer, pus which has formed in, or been admitted into the veins which terminate in the vena portæ, leads to metastatic abscesses in the liver, while pus in the veins of the general circulation commonly leads to abscesses in the lungs. The explanation of this fact is this: in the first instance, the pus globules are arrested by the capillaries of the liver, which are the first capillary vessels they attempt to pass, while in the second instance, the same fact is true of the capillaries of the lungs. It is stated, also, that the form of abscess known as metastatic is commonly found in the lungs of those who die of glanders.

The influence of external wounds in the production of pneumonia, is very limited. Every one must have observed that the common accident of fractured ribs, seldom leads to the production of this disease. It is also the opinion of army surgeons, that wounds of the lung, if they do not implicate any of the great vessels of the organ, are not attended by much danger from the supervention of inflammation. I may remark, however, that foreign bodies lodged in the lungs, especially such as enter by the mouth, create a good deal of secondary inflammation. Indeed, they gradually lay the foundation for an abscess in the lung, which may, even after a long interval of time, lead to a fatal termination.

In attempting to estimate the *mean duration* of pneumonia in the healthy adult, you will probably be near the truth, in fixing the period at about two weeks. Yet cases vary so much, that you may find the disease terminating in a week, or you may see it protracted to five or six weeks. I refer now to the duration of the disease in cases which recover, and limit the disease to the period of full convalescence, as established by Louis for

acute diseases, viz.: the period when the patient is able to sit up during the greater portion of the day, and to eat solid, nutritious food with a good appetite, and when he is entirely free from febrile excitement. Yet, if you examine such cases, you will find that all traces of the disease have not disappeared. The patient still coughs and expectorates a little, and on examining the chest you will still find the physical signs of the disease; a slight dulness on percussion, a feeble respiration, and perhaps a subcrepitant rattle. Indeed, some degree of congestion still remains, which slowly passes away, perhaps only after several weeks, when the lung regains entirely its natural condition.

On the other hand, cases which terminate fatally, generally do so about the tenth day, although, as in those cases which recover, the duration is subject to wide limits. I speak now of the primitive pneumonia of the adult.

In new-born children, Valleix found the disease of short duration, usually terminating fatally during the first week, often, indeed, in two or three days. Rilliet and Barthez, in their observations on children from one to fifteen years of age, found the disease exceedingly variable in its duration. In the primitive form, its general duration does not differ materially from what we notice in adults—ten to fifteen days. In the secondary form its duration varies from five to twenty-five days, and it is probably longer in its duration in those cases where it occurs in the course of a chronic affection, as diarrhœa, than where it occurs in the course of an acute, or febrile attack.

The *mortality* in pneumonia has been established only from the experience of Hospital practice. In estimating this question, you must recollect that there are many unfavorable circumstances in Hospital cases, which must render the mortality greater than in private practice. The previous habits of Hospital patients, the length of time which often elapses before any efficient mode of treatment has been adopted, the frequent exposure to unfavorable hygienic influences after the disease has commenced, both at home, and during a removal to the Hospital, no doubt operate unfavorably upon so severe a disease, and materially increase its mortality. It must be evident, also, that the disease will be more frequently fatal when it occurs as a complication, than in

its primitive form. Occurring in the course of acute diseases, it aggravates their severity, and often leads to a rapidly fatal termination, while in chronic diseases, the fatal issue is still more certain.

Primitive pneumonia in new-born children terminated fatally in all the cases reported by Valleix, but the number is too small to establish any definite conclusion, being only three. The cases reported by Rilliet and Barthez, occurring in children from one to five years of age, are more numerous—twenty-four; of these, three died, making the mortality one in eight. While in older children, from six to fifteen years of age, the mortality is still less. Among forty cases observed by Gerhard, only one terminated fatally, while in one hundred and sixteen cases between the ages of sixteen and thirty years, eight terminated fatally, or one in thirteen. After the age of thirty years, the mortality seems gradually to increase. Thus, from thirty to forty years, the mortality is about one in seven; from forty to fifty years, one in six; from fifty to sixty years, one in five; while of those over seventy years of age, at least four in five terminate fatally.

The influence of age, therefore, on the mortality of primitive pneumonia, is a fact very clearly established. The period of life during which you may expect the most favorable termination is before the age of thirty years. After this period, the disease evidently becomes more fatal, without our being able to refer this to any other cause than to the progress of age. In young children, this primitive form of the disease, it will be observed, does not appear to be more fatal than in adult life, but then it is comparatively rare, most of the cases of pneumonia at this age being secondary, and much more fatal.

Secondary pneumonia, occurring in the course of a variety of acute and chronic diseases, will influence the mortality of these diseases according to the circumstances of the original disease, the extent of the secondary affection, and the time of its appearance. But in all cases, it is a very serious, indeed, usually a fatal complication. Compare, for instance, the mortality of the primitive pneumonia in children, with the mortality of secondary pneumonia, observed in those of the same age by Rilliet and Barthez at the Children's Hospital at Paris. In eighty-one cases in which pneumonia existed as a complication, seventy-

seven terminated fatally. When pneumonia occurs as a complication of measles, it is very fatal, twenty times in twenty-one cases. Rilliet and Barthez, however, make the mortality less: when the pneumonia is the only disease complicating measles, the mortality is only one in three or four; while if the measles have occurred in those suffering from anterior disease, the mortality is at least double. Typhoid fever is another form of disease very frequently complicated with pneumonia. Louis found it in seventeen of forty-six fatal cases among adults, and Rilliet and Barthez noticed it in twenty-two cases of typhoid fever, or in about one-fifth of the cases. Of these, the disease was fatal in eighteen cases.

In chronic affections, especially in chronic diarrhœa, in cancerous and tuberculous disease, pneumonia is very apt to occur, and hurry the patient rapidly out of existence. It has been observed, however, in young children, that the disease often assumes a chronic form, passing slowly into hepatization, and that five or six weeks even may elapse before the fatal issue.

It is thought that females more frequently die of the disease than males.

Intemperate persons are apt to be seized with the symptoms of pneumonia, followed by those of delirium tremens. This complication has very frequently a fatal termination.

It is remarkable, that tubercles in the lungs do not appear to aggravate an attack of pneumonia, provided the original disease is not far advanced. In the advanced stage, however, of tuberculous disease, an intercurrent pneumonia often hastens the fatal termination of the case.*

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* Proportion of deaths from Pneumonia to the whole population:

Boston,	1 in 1,110
New York,	" 557
Baltimore,	" 15,364
Charleston,	" 2,378
London,	" 478

Proportion of deaths from Pneumonia to the whole number of deaths:

Boston	(period of	5	years),	1 in 44 deaths.
New York,	"	2	"	1 in 17 "
Baltimore,	"	4	"	1 in 332 "
Charleston,	"	5	"	1 in 69 "
London,	"	1	"	1 in 11 "

It is to be presumed, that the reports from Baltimore are far from being correct,

LECTURE VII.

PNEUMONIA.

Symptoms of primitive pneumonia in its different stages in the adult.—Double pneumonia.—Pneumonia of children.—Secondary pneumonia.—Appreciation of the more important symptoms and physical signs in the diagnosis of pneumonia.

PRIMITIVE pneumonia, in the adult, is usually ushered in by a chill, followed by febrile reaction, and, in some cases, by the precursory symptoms of languor, pain in the head, back, and limbs, and loss of appetite; symptoms to which the French have given the expressive term *malaise*. These general symptoms

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the certificates of death being signed by the sextons, and not by the attending physician.

The number of deaths from Pneumonia, during three successive years, in the city of New York, was:

Males, 1384; Females, 1174: Total, 2558.

The number of deaths from Pneumonia, during three successive years, in the city of New York, was:

From December to June,	1665
From June to December,	893
Total,	2558

In London, the number of deaths from Pneumonia, during one year, was:

From December to June,	2242
From June to December,	1681
Total,	3923

The proportion of deaths from Pneumonia at different ages, and during three successive years, is as follows:

	1 yr.	1 to 2 yrs.	2 to 5 yrs.	5 to 10 yrs.	10 to 20 yrs.	20 to 30 yrs.
1848	193	101	101	23	33	55
1849	258	112	139	52	23	79
1850	240	139	137	58	21	72
	691	352	377	133	77	206

	30 to 40 yrs.	40 to 50 yrs.	50 to 60 yrs.	60 to 70 yrs.	70 to 80 yrs.
1848	68	52	43	17	11
1849	85	63	39	41	26
1850	82	57	51	37	22
	235	172	133	95	59

In persons above eighty years, there were nineteen deaths from Pneumonia.

are soon followed by pain in the affected side, by dyspnœa, and by cough. Called to the patient soon after the attack, you will find him in bed, and usually upon the back, with the face flushed, the skin hot, the pulse accelerated, the tongue coated white, the appetite impaired, the thirst considerable, the bowels constipated, and the quantity of urine diminished. He complains of pain in the head and limbs, but especially of an acute pain in the lower portion of the thorax. His cough is more or less urgent and painful, his expectoration often trifling, but frequently viscid, and sometimes rusty in appearance. On examining the chest, on the side where the pain exists, you will notice a slight degree of dulness over a limited portion, most frequently below the inferior angle of the scapula, the respiratory murmur feeble, and not unfrequently the crepitant rattle, especially during a full inspiration. Over the remainder of the lung nothing unnatural is observed, nor on the opposite side, unless that, in this latter portion of the chest, the respiratory murmur is rather more distinct than common, from the increased activity of the unaffected lung. These symptoms and physical signs indicate the first stage of pneumonia.

But these conditions seldom remain long without new evidences of disease. The constitutional and rational symptoms, indeed, may remain unaltered, except, perhaps, increased in degree, but in a short period of time, usually as early as the second or third day, you will find the dulness on percussion more marked and extended, and a bronchial respiration taking the place of the feeble murmur, and of the crepitation; evidences that the second stage, or that of hepatization, has formed. With this existence of the second stage in the portion of lung first affected, you will have the evidences of the first stage in the immediate neighborhood. Indeed, for a time, the signs of the two stages may be blended in the same portion of the lung; you will hear in the same spot a bronchial respiration and a crepitation. With these evidences of an increase in the local disease, you will naturally expect to find an aggravation of the rational symptoms, and especially of the dyspnœa; while the constitutional symptoms undergo an equal increase of severity, the pulse becoming more accelerated, the fever more considerable.

In the progress of the case, the physical signs gradually increase in extent, without changing, materially, in their character. The dulness increases, both in degree and in extent, until often the whole posterior, lateral, and lower half of the affected side, is affected. The bronchial respiration extends also, becomes more blowing, and loses all evidence of crepitation, although in the outskirts of the dulness and of the dry, blowing respiration, a crepitus may still, not unfrequently, be heard. The respiratory murmur in that portion of the affected lung where there is no evidence of disease—no dulness, no crepitation—is feeble, as if that portion of the lung was crippled in its action by the neighboring disease.

The rational symptoms also undergo important changes. The dyspnœa gradually increases, but the pain subsides, and the expectoration, although still viscid, has more of the character of bronchial mucus, and has probably lost all appearance of rustiness. During this progress, the pulse continues accelerated, and often increases in frequency, the skin continues hot, the secretions are diminished, and the tongue becomes more loaded with a dirty white coat. These signs and symptoms mark the height of the disease in ordinary well-developed cases of primitive pneumonia which are destined to have a favorable termination. They indicate an hepatization of one-half, or of two-thirds of one of the lungs, which, in a majority of cases, perhaps, reaches its full development about the tenth day of the disease.

The disease continues, apparently stationary, for a day or two, and then the period of resolution may commence. Sometimes the evidence of this favorable change is first observed in the physical signs; sometimes in the rational and in the constitutional symptoms; but frequently the evidences of an improved condition are manifested in all of them at the same time—the pulse becomes less frequent, the febrile excitement abates, the dyspnœa becomes less urgent, the cough and expectoration more easy. While examining the lungs, especially the portion last affected, you will notice the crepitant rattle returning again where the dry, blowing respiration existed, larger and more moist than in the earlier period of the disease. Gradually the dulness diminishes, the bronchial respiration gives place to a feeble mur-

mur, harsh, and mixed with coarse crepitation, until, after the lapse of five or six days, the period of full convalescence is reached. Still, however, the patient coughs and expectorates bronchial mucus; still some degree of dulness remains; and the feeble respiration, mixed with crepitating bubbles during inspiration, or masked by a mucous rattle, indicate that the lung has not yet returned to its natural condition: it is still congested. But gradually these evidences of pre-existing inflammation subside, and the lung regains its natural condition.

When, on the other hand, the disease tends to an unfavorable termination, that is, to suppuration, the constitutional symptoms do not abate; the dyspnœa continues, or becomes more aggravated, expectoration is more difficult, and occasionally presents the appearance of a blackish or dark-red diffluent secretion, like the juice of preserved prunes; the dulness on percussion continues, and, mixed with the bronchial respiration, you will begin to hear a rattle, at first rare, soon becoming more abundant, and resembling a mucous rattle—these are the indications of suppuration; and the patient gradually dies, not so much from exhaustion, as from oppression. The pulse becomes, indeed, more accelerated and feeble—sometimes it is irregular—but it is the dyspnœa, the inability to expectorate, the rattling of mucus in the trachea, which more especially mark the fatal issue. The intellectual faculties are usually preserved until near the final scene, when delirium, and even coma, may ensue.

Most cases which terminate fatally, do so without presenting any conclusive evidences of suppuration. Many indeed die, without the disease having advanced beyond the stage of hepatization, or with only a slight tendency to the suppurative stage, indicated on post-mortem examination by a grayish or yellowish tint with a little more softening than attends hepatization, but without any marked alteration in the physical signs in the lung, or any evident change in the rational or constitutional symptoms. If, however, you notice a coarse, moist crepitation passing into a mucous rattle, and coinciding with an increase, instead of a diminution of the rational and the constitutional symptoms; if the prostration increases, the pulse becomes more accelerated, if sordes collect about the teeth, and the tongue

becomes dry, while the dyspnœa becomes more urgent and the expectoration more difficult; and especially, if the prune-juice sputa appear, you will have reason to believe that suppuration has taken place. Still, it must be remembered, that even these conditions may exist, and yet the lung be only in the stage of hepatization. I think, however, that such cases are rare.

The direct tendency there is in pneumonia to suppuration would undoubtedly lead more frequently to abscess in the lung, did not the patient die before this can be accomplished. Hepatization alone, in primitive pneumonia, is quite enough to destroy life in persons of ordinary vigor, when the disease has affected a considerable portion of the lung, as, indeed, is most frequently the case. But now and then, when the pneumonia is limited, and most frequently in those who are of feeble constitution, an abscess is formed, or several abscesses may exist at the same time. The patient may die without any suspicion of abscess during life. But if the case is unusually protracted; if, after a temporary improvement, hectic symptoms supervene; if the patient suddenly expectorates a considerable quantity of pus, then the existence of an abscess is sufficiently probable. With these well-marked rational and constitutional symptoms, however, you may be able to derive little or no advantage from physical signs. As in the case of tuberculous abscess, a cavity full of pus not communicating with a bronchial tube, may exist in the centre of a condensed pulmonary tissue, and give no evidence of its existence. It is only when the cavity is evacuated by a bronchus, that the signs of a cavity become distinct. If, indeed, the opening into the bronchus be small, you may fail to hear a cavernous respiration, but you will probably detect a gurgling, and the chinking sound on percussion. These abscesses usually wear out the patient, and may terminate life after an interval of about two months from the first attack. I remember a lady about thirty years of age, of delicate constitution, who became affected with pain in the left side, followed by dyspnœa, a trifling dry cough, and a very rapid pulse. She was feeble, and confined mostly to the bed. On examination, I found the lower lobe of the left lung very dull on percussion, and the respiratory murmur nearly, or quite, absent. I believe, I

thought, that egophony also existed. At any rate, I regarded the case as a subacute pleurisy, a form of disease which often ushers in the symptoms of tubercles. This impression seemed to be confirmed, for the patient did not improve, but, on the contrary, had hectic symptoms, and, indeed, all the usual symptoms of tuberculous disease, the physical condition of the lung remaining unaltered. After a time, however, a month or two, she began to improve, and continued to improve most decidedly in her rational and constitutional symptoms. From being apparently near her death, she was able to ride several miles. Unfortunately, she was suddenly seized with cerebral symptoms, followed by fatal convulsions. On post-mortem examination, the lower lobe of the left lung adhered firmly to the chest. Its tissue was compressed as you see it in ordinary pleurisy. In the centre of the lung was an abscess as large as a hen's egg, invested by a firm, organized membrane, full of healthy pus, not communicating with the bronchi. The remaining portions of the lungs were healthy. There were no tubercles. Several recent abscesses were formed in the substance of the brain. This case I did not attend regularly, but I saw the patient at several different times, in consultation.

Double pneumonia sometimes occurs in the primitive disease of the adult, the form I am now considering. One lung is first affected, and in the usual manner. During the progress of the case, the second lung becomes involved, and was it not for auscultation, you would hardly be able to detect its existence, for no new symptoms are usually added to those already existing. Even pain in the newly affected side does not generally exist to warn you of the new danger, and although the constitutional symptoms and the dyspnœa may become aggravated, yet this might happen from an extension of the disease in the lung primarily affected. But by auscultation, you will detect the same phenomena developing themselves as in the lung first affected, the same dulness, feeble respiration, crepitation, and finally, the bronchial respiration. When both lungs are thus crippled by disease the constitutional and rational symptoms often become more marked, and the mortality is increased.

The *primitive pneumonia of children* under six years of age,

is a rare form of disease. When it does occur, it possesses the same anatomical characteristics as the disease in adult life. It usually affects but one lung, and particularly the inferior and the posterior portion, and it spreads over a continuous surface. It is an acute disease, with the symptoms of high febrile reaction, and with well-marked rational and physical signs. The latter, as you might expect, do not differ from those noticed in the adult; except, perhaps, that the crepitant rattle is less frequently heard. The symptoms, however, present some peculiarities. The child, when young, does not often expectorate, and the sputa which are expectorated are less frequently rusty than in the adult. The nervous system is much more decidedly affected. At the onset of the disease even, a good deal of agitation may be present, and, indeed, violent convulsions may ensue. Towards the close of life, in fatal cases, the tendency to coma is sometimes very decided.

Primitive pneumonia is not an infrequent form of disease in *new-born children*. It is usually a rapidly fatal disease, terminating during the first week of its existence, or even sooner. Its anatomical characteristics are those of the primitive pneumonia at other periods of life, a lobar or continuous consolidation of the lung, or hepatization. Its symptoms are dyspnœa, cough, and febrile excitement. Its physical signs are, dulness on percussion, bronchial respiration, and, sometimes, a subcrepitant or crepitant rattle. In this rapidly fatal disease, the febrile excitement is often of very limited duration, giving place to the symptoms of collapse—coldness of the surface, and a slow and feeble pulse.

The *secondary pneumonia* of children under the age of six years is almost always of the *lobular form*. It is almost always *double*. In these respects it differs entirely from the primitive form which I have just described.*

The mode of attack is also different. The first symptoms are those of bronchitis; viz., the same moderate febrile reaction, dyspnœa, cough, and the rhonchi usually noticed in that disease.

* In 58 cases of primitive pneumonia in young children, under six years of age, lobular pneumonia existed in only three cases.

With these symptoms, however, you will soon perceive a subcrepitant rattle at the base of both lungs, which is the exception to the general rule in the acute bronchitis of adults. But in the progress of the case, the fever and the dyspnœa increasing, and the cough becoming more painful, you will be able to detect the development of dulness on percussion, at first, slight in degree and doubtful, over the posterior and probably the inferior portion of the lungs. But as lobule after lobule becomes inflamed, a bronchial respiration will be heard, indistinctly, perhaps, owing to its imperfect development, and to its being masked by the subcrepitant rattle, which is usually abundant. In a short time, however, the dulness on percussion becomes more decided, the bronchial respiration more distinct, as more lobules become hepatized, and as the subcrepitant rattle diminishes, to give place, perhaps, to the finer and less masking crepitant rattle. These signs of a gradual consolidation of the lungs are almost always, like those of the preceding bronchitis, *double;* that is, they exist in both lungs, a circumstance which tends to obscure the physical diagnosis, especially in the early stage of the disease, when a comparison of the diseased with a healthy lung is so important.

When this form of the disease is fully developed, a double pneumonia is easily detected by the dulness on percussion, by the bronchial respiration over the posterior portions of the lungs. The dyspnœa is great, the alæ nasi distended and in constant motion; the cough painful; the face flushed at times, at other times pale and livid. There is an accelerated pulse, and much constitutional fever. In cases which terminate favorably, the progress of resolution does not differ, materially, from that noticed in the primitive or lobar form of the disease, except that it is more slow and more irregular. The dulness on percussion diminishes, the bronchial respiration becomes less distinct, the subcrepitant rattle more abundant. The physical signs assume again the characteristics of bronchitis, and finally disappear.

In both the primitive and the secondary pneumonia of young children, death is preceded by the same symptoms. The countenance becomes pale, the lips livid, the respiration much op-

pressed, and often irregular; the pulse very feeble, accelerated, intermittent; and finally, coma supervenes.

Secondary pneumonia, occurring in the course of acute diseases, as continued fever, the eruptive fevers, is modified in its constitutional symptoms by those of the original disease. The rational symptoms are apt to be more or less latent, being masked by those of the primary affection. Thus pain in the chest may be absent, the cough trifling, or not differing from that noticed in the original disease, and the expectoration, as indeed not unfrequently happens in the primitive form, destitute of the characteristic appearance. It is to auscultation that you must turn to obtain the knowledge that a secondary pneumonia exists. An aggravation of the general symptoms, some imperfectly developed rational symptoms, may lead you to suspect its existence. But you have only to examine carefully the chest, at least in most instances, to find your suspicions confirmed, when the disease is actually developed. It is in continued fever, in measles, in small-pox, and in croup, among the acute diseases, that you will have most reason to apprehend the secondary development of pneumonia.

The secondary pneumonia which occurs as a complication in chronic diseases, is modified by the state of the constitution as affected by the original disease. When chronic disease has made but little inroad upon the general health, pneumonia may occur, and will differ in no material respect from the primitive form. This is true even of the pneumonia which occurs in the course of early tuberculous disease of the lungs. When the disease occurs in patients in whom a pulmonary disease of long standing has existed, and in whom much disturbance of the circulation has long existed, it is apt to assume an aggravated form, particularly in reference to the dyspnœa. You will observe this in the pneumonia that complicates emphysema of the lungs, and in the cases which occur in heart disease. Finally, the pneumonia which occurs in chronic disease, the seat of which is remote from the lungs—chronic diarrhœa, or cancer of the womb, for instance—and in which the vital powers are at a very low ebb, the disease is almost entirely latent, and is generally rapidly fatal. You will find your patient sinking without any decided symptoms that

can be referred to the lungs; there is a little cough, perhaps, and dyspnœa, and slight febrile reaction. But these are often very slight. In such cases, if you examine the lungs you may detect the physical evidences of pneumonia, probably, in the first or congestive stage—the disease terminating fatally before much hepatization has occurred. When the pneumonia which complicates the advanced stage of chronic disease assumes the lobular or disseminated form, as is most frequently noticed in children from one to six years of age, it is often exceedingly slow in its development, continuing for five or six weeks before it reaches a fatal termination. A trifling cough may be noticed, a little dyspnœa, perhaps slight febrile excitement; while gradually, a dulness on percussion and a bronchial respiration are developed, as lobule after lobule becomes affected and pass into the stage of hepatization, until at length the patient sinks, gradually exhausted.

I will now ask you to examine more carefully and in detail the most important symptoms of pneumonia.

The *cough* is usually one of the earliest symptoms, and it continues after the patient has passed into a state of convalescence. It is, usually, dry and moderate, at the onset, in simple cases, but is soon attended by expectoration; and this condition continues until the close of the disease. It is ordinarily suppressed and painful during the active stage of the disease, but gradually loses this character, especially in favorable cases. The short, suppressed, painful cough in no way distinguishes the disease from pleurisy, but it does serve to distinguish it from bronchitis, in which the cough is usually prolonged and sonorous. This fact is of great importance in distinguishing the bronchitis of young children from the more severe inflammatory affections of the lungs.

The *expectoration* is of much more importance than the cough in the pneumonia of adults. In children, it is of comparatively little importance, as it is not often characteristic, and is usually swallowed. But in adults, often as early as the second day, it becomes semi-transparent, viscid, and rusty, with minute, equal-sized air-bubbles. After a few days, it often assumes a yellowish hue, and finally, sometimes, a greenish aspect. The yellow-

ish tint is undoubtedly derived from the admixture of a very small proportion of blood, less than that which produces the rusty appearance. Sometimes, the characteristic rusty or yellowish sputa are abundant, and constitute the mass of the expectoration. At other times, the secretion is principally bronchial mucus, a few specks of the rusty expectoration only being apparent. Or, again, there may be no rusty expectoration, but the bronchial mucus is more viscid than it is in simple bronchitis. The quantity of characteristic expectoration has no connection with the extent of the disease. It may be abundant, when the disease is limited; or absent, when the disease is considerable, —its quantity being very much dependent on the ease with which it is expectorated. It comes from the air-cells, and marks, I think, the passage of the first stage of the disease into the second stage; so that when the affected lung has passed into hepatization, or when, from the extent of the disease, or from the exhaustion of the patient, matter can no longer be expectorated from so remote a source as the air-cells, you will find no longer the rusty, characteristic expectoration. It seldom, indeed, continues after the seventh or eighth day from the attack, when it is succeeded by a viscid and opake bronchial mucus. In certain rare cases, we have an expectoration towards the close of the disease of a thin, diffluent, reddish, or even blackish fluid, resembling the juice of preserved prunes. It contains but little air, although a slight froth may be noticed on its surface. This is usually a fatal sign, and is generally found connected with the third, or suppurative stage of the disease. But as cases in which it has occurred have been known to recover, it may possibly occur independently of the third stage, or when this stage is only commencing its career.

Dyspnœa is a constant and early symptom of pneumonia. The cough may not exist, or only appear after the disease is developed, the characteristic expectoration may fail entirely; indeed, this is not very infrequent, but dyspnœa is a constant symptom. Unfortunately, it is not characteristic, as it occurs in all acute and chronic affections of the lungs. In the primitive pneumonia of the adult, the inspirations are more frequently about thirty in a minute. In simple, well-marked cases, dur-

ing the height of the disease, they may reach forty in a minute, and in extreme cases, especially when the pneumonia is double, you may count fifty or even sixty inspirations in a minute. In children, of course the respiration is more rapid, forty to fifty inspirations a minute being common, and seventy or eighty inspirations not very rare. In old people, the respiration, other things being equal, is usually less accelerated than in those of middle life. It is dyspnœa which most frequently marks the supervention of pneumonia in its secondary form.

Pain in the chest belongs equally to pneumonia and to pleurisy. It has the same origin, the same seat, the same general duration. It has been thought that the pain was commonly more severe in simple pleurisy than in pneumonia. Pure pneumonia, that is, when it is central and uncomplicated with pleurisy, is not usually attended by pain. There will be a sense of heat and oppression in the chest, but this is very different from the acute stitch which occurs in cases in which the pleura is inflamed simultaneously with the lungs. In a majority of cases, the pain is felt in a circumscribed spot just below the nipple, or at the base of the chest laterally, or in front. It indicates the side affected, but it does not serve to locate the precise seat of the disease. Thus, in pneumonia of the upper lobe, the pain is usually felt in the lower portion of the chest. When the pneumonia is double, the side last affected is seldom the seat of pain, and the same absence of pain is observed in cases of secondary pneumonia.

The pain in pneumonia, like that in pleurisy, is uniformly increased by a full inspiration; there is also a degree of tenderness on pressure. When not severe, the pain sometimes extends over a considerable surface. Indeed, the seat of the pain under the nipple marks the spot where the expansion of the chest is greatest, and of course the spot where the inflamed pleura is more easily put upon the stretch. The circumscribed seat of the pain indicates that this stretching is at once arrested, else no doubt the pain would extend to other portions of the chest, as it may do when, not being very severe, the expansion of the chest is not arrested.

From the statements I have just made, it will appear that

dyspnœa is the most constant of the rational symptoms of pneumonia, while it is the least characteristic. This may be said also of the cough, which is seldom absent, while the pain, less constant, is found to possess the same characters as in simple pleurisy. Indeed, the pain in both cases is a pleuritic pain. In certain cases, pain attacking the muscles of the chest can hardly be distinguished by its local characteristics from that of pleuritis. It is apt, however, to be more diffused, to be attended by more tenderness on pressure, and to be less fixed in its seat. It is not at all uncommon, however, for the pain in both pneumonia and pleurisy to be wandering in its character at the onset of the disease. It may even present itself below the region of the chest, as low as the iliac region. In one instance I noticed the pain on the opposite side of the chest from that affected with pneumonia. At all events, the pain existed without any physical evidences of pneumonia on the same side, while on the opposite side these evidences were distinct. The rusty and viscid expectoration, is the true characteristic rational symptom of pneumonia, and if it exists in ever so minute a quantity, it stamps the disease; but unfortunately it is limited in its duration, and is, not unfrequently, entirely absent.

It is the absence of the rational symptoms, or their imperfect development, which tends chiefly to render pneumonia a latent affection, and it is especially the previous existence of some other disease which leads to this result. In old age, also, the sympathies of the system are rendered dull, and this is a cause of latency. A natural dulness of intellect, the existence of insanity in some form, and the period of early childhood during which the sensations are not easily expressed and when the expectoration is usually swallowed, are also causes which operate in obscuring the rational symptoms.

But these difficulties in the diagnosis of pneumonia by the rational symptoms, do not apply, in any thing like the same degree, to the physical signs of the disease. If you except the rare cases, in which the pneumonia is central, and the cases in which the pneumonia is lobular or disseminated, the physical signs are well developed, and if not always characteristic, at least indicate a serious affection of the lungs.

Of the physical signs, the most characteristic is the *crepitation*. This sign, like the rusty expectoration, indicates an affection of the air-cells, the primitive seat of the pneumonia. Like the latter, it belongs to the first stage of the disease. It is a fine, dry, equal crackle, heard only during inspiration, and seeming to burst upon the ear from a circumscribed spot. Situated deep in the lungs, remote from the rushing current of air, this current may not have force enough to develop it, and then a more forcible inspiration, as that which precedes the act of coughing, may be necessary to discover it; or the stage of the disease in which it exists may have nearly, or quite, passed away, and then it, of course, will not be heard, or only feebly and at intervals. When resolution commences, it returns, but more coarse and moist than before, and it gradually assumes the characters of a subcrepitant or of a mucous rattle.

In certain cases, in which much bronchitis exists, the subcrepitant rattle either masks the crepitation or takes its place. Situated in the smaller bronchi, it possesses a mixed character. It is more fine, more equal, but less moist than the well-developed mucous rattle, but like it, is heard during expiration as well as during inspiration. Thus the complication of bronchitis modifies the crepitation precisely as it does the expectoration.

The influence of pneumonia in its first or congestive stage, is to render the respiratory murmur feeble, but as soon as hepatization occurs it becomes *bronchial*, or *blowing*. This bronchial respiration occurs early in the disease, often as early as the second day, and continues until the period of resolution, or until death. When perfectly developed, it is dry, but in many cases it is mixed with some crepitation, indicating that the part affected is passing from the first into the second stage of the disease; but when the second stage is fully formed, it is dry, blowing, and equally distinct during inspiration and expiration. If you watch its progress, you will find that the expiratory murmur first becomes prolonged, then blowing, and that the inspiratory murmur is, afterwards, similarly affected. This blowing respiration is not characteristic of pneumonia. It occurs in other cases: sometimes, as I shall have occasion to remark, in

pleurisy, in tuberculous disease; in speaking of dilatation of the bronchi, I have already mentioned its occurrence.

Dulness on percussion attends every stage of the disease. Slight and limited in the early stage, it becomes decided and, usually, extensive, when hepatization is fully developed. It is in central pneumonia, and still more frequently in the lobular variety of the disease, that the dulness is least marked and most slowly developed. In these cases, as, indeed, in the ordinary form of the disease, it keeps pace with the development of the bronchial respiration.

In cases of primitive pneumonia, it is common to find the dulness on percussion and the bronchial respiration gradually extending until they occupy one-half, or even two-thirds of the surface of the lung affected. In certain rare cases, the whole lung may have become consolidated. Such cases are usually fatal.

Whenever the lung is consolidated by inflammation, an increased resonance of the voice accompanies its development. Slight, in the stage of congestion, it is very marked when the stage of hepatization has formed, constituting what is called bronchophony.

When the form of the disease known as splenization occurs, the physical signs differ, in some respects, from those noticed in hepatization of the lung. With dulness on percussion, you may notice a more or less complete absence of all respiratory sounds. You may, perhaps, notice a coarse, rare crepitation. This absence of respiratory sound may, it is supposed, occur, however, in cases of very marked hepatization, either from the compression of the bronchi by the surrounding lung, or from a feebleness of the respiratory powers. Such a case is, however, very rare, if indeed its authenticity is, as yet, well established.

I will remark, in concluding this portion of the subject, that the development of the physical signs of pneumonia may occur over any portion of the pulmonary surface. As a general rule, it is the posterior portion that is affected; to this rule there are but few exceptions. Less constantly, but still very frequently, it is the inferior portion that presents the first signs of the disease. In clinical practice, then, when pneumonia is suspected,

your first and most careful search for the physical signs should be at the base of the lungs, posteriorly. It is at the two extremes of life that the superior portion of the lungs is most frequently found to be first attacked by the disease.

LECTURE VIII.

PNEUMONIA.

Constitutional symptoms.—Pulse.—Digestive organs.—Jaundice.—Bilious pneumonia.—Cerebral symptoms.—Chronic pneumonia.—Treatment.—Expectant method.—Blood-letting.—Tartar-emetic.—Blisters.—Expectorants.—Treatment of typhoid pneumonia; of secondary pneumonia; of the pneumonia of children.

At the close of my last lecture I called your attention to the rational symptoms and to the physical signs of pneumonia. The *constitutional symptoms*, as they differ in no material respect from those noticed in other inflammatory affections of important organs, need not, now, occupy much of your time. It may be said, however, that the febrile reaction in this disease is, usually, considerable, and sometimes intense. It is in those far advanced in life, in those worn out by chronic disease, and in those of a naturally delicate and feeble constitution, that this febrile reaction is less manifest.

The pulse, in this disease, is, generally, full and soft, and ranges from ninety, to one hundred and ten in a minute. I think that one hundred and ten is a very frequent number for the pulse in primitive pneumonia of ordinary severity, in an adult patient of good constitution. It is not uncommon, however, for the pulse to reach one hundred and twenty pulsations in a minute, and even this need not be regarded with apprehension. When, however, it much exceeds this number, reaching to one hundred and thirty or one hundred and forty pulsations in a minute, it is a more serious affair. My experience has taught me that, in pneumonia, a pulse exceeding one hundred and

twenty, is not free from danger to the patient. In fatal cases, the pulse becomes feeble and much accelerated, sometimes it is irregular. I have not unfrequently noticed, however, that the pulse continued full and strong until near the close of life.

In children, of course, the pulse is more accelerated than in adults. A pulse of one hundred and twenty in a minute, indicates no unusual severity in the disease. It sometimes reaches one hundred and fifty and one hundred and sixty, or even a higher number. In fatal cases, it is apt to be exceedingly rapid before death.

The condition of the digestive organs commonly presents no important peculiarities. The tongue is coated, sometimes it is very much so; the appetite is gone, the urine is scanty and high-colored. Generally, there is no evident biliary derangement. But, sometimes, in the course of the disease, the skin and eyes become yellow, and the urine, and even the expectoration, become affected in the same manner. There is, evidently, bile in the system. But in these cases, there is no evident disease of the liver, and nothing indicating organic changes is found after death. There seems to be a partial want of the secretion of bile by the liver—not a complete stoppage of the secretion, for the stools may continue to be colored by it, as is usual. It has been thought, and this seems to be the fact, that this complication is more common when the lower half of the right lung is the seat of inflammation; but there are striking exceptions to this rule.

A form of pneumonia called *bilious*, is sometimes met with, especially in miasmatic regions, in which a redundancy of the biliary secretion is the leading characteristic. In these cases, the skin may, indeed, become yellow, and the urine also; but the intestinal canal and the stomach are likewise loaded with the secretion of bile. A bitter taste in the mouth, bilious vomiting, a tender and full feeling at the epigastrium, sometimes free bilious discharges, with a tendency to the typhoid state, are the prominent features of this form of the disease. You will see at once, that although more or less of jaundice may be present in the two forms of biliary complication to which I have alluded, yet that they differ very essentially in their nature.

It is not uncommon for a patient to vomit at the commence-

ment of the disease, especially during or after the early chill. I think this is common in most febrile affections; but any permanent vomiting, and, especially, any other symptoms indicating gastritis, are rare; unless, indeed, the patient has been badly treated, or has labored under the disease before the attack. Neither do I think diarrhœa of common occurrence in the primitive form of the disease. In children, perhaps, this is more frequently noticed.

In a primitive pneumonia, although there may be more or less pain in the head at the onset, and during the acute stage of the disease; although, at the closing scene, in fatal cases, the intellect may sink into coma and low delirium—yet, as a general rule, the intellectual powers are remarkably sustained in this disease. The coolness and composure which mark its progress, like the sanguine expectation of recovery which belongs to phthisis, is to me one of its most striking and pathognomonic features. This composure continues often until the close of life, or until near this period, when a slight wandering of the intellect, or a tendency to coma may supervene, as, indeed, happens in most diseases. But there are some striking exceptions to this general rule. Nervous, hysterical females may be attacked, even early in the disease, with cerebral symptoms which will alarm you very much unless you understand their true character. They are entirely independent of organic disease of the brain, although, possibly, they may lead to inflammation, and they generally subside *pari passu* with the pneumonia. I will relate to you a striking case of this kind. A young married lady, of delicate constitution and of a highly nervous temperament, was attacked with the symptoms of pneumonia affecting the lower lobe of the left lung, which was in the stage of hepatization. The fever was moderate in degree, but the pulse was 136 in a minute. On the fourth day from the attack, she passed a restless and agitated night. The next morning her face was flushed, and her head hot. Her eye was bright and wild, her answers were impetuous, and her movements rapid and sudden; but she was quite sensible. During the day, furious delirium ensued, during which she complained that her head was very hot. The next morning she appeared nearly rational, but was evidently much excited.

She complained of pain in the head; her face was flushed and hot, and she was frequently picking at the bed-clothes. The febrile excitement was moderate; the pulse was 120 a minute, and soft. The rational symptoms of pneumonia were very indistinct. There was but little cough or acceleration of the respiration; but the physical signs of a limited pneumonia at the base of the left lung continued unchanged, viz., dulness on percussion with bronchial respiration. During the course of this day, the symptoms of cerebral excitement suddenly changed their character. The patient became comatose; the pupils were insensible and much dilated; there was a tendency to sinking down in the bed, a tracheal rattle, and a feeble pulse. The free use of stimulants, however, soon removed these most alarming symptoms. The symptoms of cerebral excitement again returned, but in a milder form. There was occasional delirium, and a total absence of sleep. But on the tenth day from the attack, the pneumonia passed into the stage of resolution, and with this change the cerebral symptoms rapidly disappeared, and the patient speedily recovered. This case was treated as an ordinary case of pneumonia, except that leeches were applied to the head, and opium, at times, was freely administered. But the most relief to the cerebral symptoms was obtained by the application of ice to the head.

Intemperate persons, also, are an exception to the general rule. It is not unusual in them for the symptoms of delirium tremens to ensue; and this complication aggravates decidedly the prognosis of the case. When decided and continued delirium ensues in persons not of a nervous temperament, of regular habits, and without any moral cause to account for its existence, it is a very serious symptom in pneumonia, passing more or less rapidly into coma and death. Under these circumstances you may expect to find evidences of inflammation in the brain, especially in the membranes of the organ. In children, a more frequent disturbance of the nervous system is observed. An attack of pneumonia is frequently ushered in by great agitation, and even by convulsions, which may subside, or be succeeded by more or less delirium, and, finally, by coma. In the aged, also, in whom there is a tendency to the typhoid state, more or less wandering

of the intellect is by no means uncommon. In both extremes of life, these disturbances of the brain are not usually attended by any marked organic lesions.

Chronic pneumonia is so rare a form of disease, that I know nothing positive of its symptoms. I have never met with a single recorded case. Laennec states that he had noted some cases, but that they had been mislaid. Andral mentions, that in this form of disease the symptoms of chronic bronchitis exist, with emaciation, and with the physical signs of dulness on percussion and bronchial respiration. But these symptoms and signs are not characteristic; they might occur in cases of dilatation of the bronchi, or in tuberculous disease of the lung. Stokes's opinion, that such cases, when cured, result in a contraction of the affected side, render it probable that he mistook cases of pleuritis for this disease.

Pneumonia being a disease the diagnosis of which is, for the most part, easy, and the progress of which can be traced by the physical signs, has been subjected to the most careful study, as to the influence of remedies upon its duration and upon its mortality.

In the first place, the experiment has been tried how far the expectant treatment—low diet, rest, and occasional laxatives, is capable of overcoming the disease. And there can be no doubt that very many cases, occurring in a mild form and in good constitutions, recover with no other treatment. M. Grisolle tried this experiment in eleven cases, in nine of which the disease had passed to the second stage. They all recovered, and the period of convalescence dated from the twelfth day of the disease.

But the success of this method is no argument in favor of its employment, even in mild and simple cases. The same observer selected thirteen similar cases, all mild in their character, and in young subjects, nine of which also were in the second stage of the disease, in which one or two general or local blood-lettings were practised, generally as early as the fourth day of the disease. These also recovered, but the period of convalescence was on the eighth day instead of the twelfth day, and the characteristic sputa ceased on the sixth day instead of the ninth day. But the

most decided influence of the treatment was upon the pleuritic pain and upon the physical signs. In the first class of cases, the pain continued, in some instances to the twentieth or twenty-fifth day, always to the seventh day, while in those who were bled, it usually ceased about the eighth day. The physical signs in the first class of cases did not begin to disappear before the end of the second week, and in some cases continued for three or four weeks; while in those cases in which blood-letting was used, the physical signs began to diminish about the seventh day, and the lung regained its permeability usually about the twelfth day.

The influence of *blood-letting* in these cases, both in diminishing the duration of the disease and in mitigating the prominent symptoms, is most clearly exhibited. Indeed, in all times, blood-letting has been regarded as a principal remedy in pneumonia; and in severe cases occurring in vigorous constitutions, it cannot be dispensed with. While the disease is in its early stage, when the skin is hot, and the pulse full and resisting, the lancet should be used freely, until a distinct impression is made upon these symptoms, especially until the pulse loses its fulness and force. No exact rule can be given in such cases as to the quantity of blood that should be abstracted. A single free blood-letting of eighteen or twenty ounces, or until some degree of faintness is induced, may reduce the pulse permanently to a sufficient degree; while in other cases, and these are frequent, the pulse may rise again in force, and require a repetition of the same practice, even for several successive times, and at short intervals. As a general rule, however, one or two blood-lettings from the arm, sufficient to induce a degree of faintness, are enough to reduce the pulse, and to prepare the system for other remedies.

Local depletion by cupping or by leeches, is to be preferred to general blood-letting when the disease is mild and the constitution delicate, even in the early stage of the disease. The cases which require this modification of blood-letting are those in which the heat of the skin is moderate, the pulse soft, and not much accelerated.

After general and local depletion, and often in connection with them, you should resort to the treatment by the tartar-emetic. This practice, as modified by the best practitioners of the present

day, consists in giving from four to sixteen grains of tartar-emetic, eight to ten grains being a medium dose, during the twenty-four hours. A grain every two hours in eight consecutive doses, allowing an interval of rest for the remainder of the twenty-four hours, is the practice I would recommend in ordinary cases of well-marked pneumonia. This is Laennec's mode of treatment. Some patients may require more, even twice or thrice this dose, if the symptoms are urgent and are rapidly progressing. Others, on the contrary, require a much smaller dose. There are many patients who require only half a grain, or even a quarter of a grain, every two hours. Every thing depends upon the circumstances of the case; and the earlier the period of the disease, the more active the symptoms, and the more vigorous the constitution, the more free may you be in the use of this remedy. In some cases, it has been given at a late period of the disease, when but little hope of any advantage could be expected, and when blood-letting could no longer be thought of. Given even in full doses, it has been attended with the most gratifying results.

The number of patients on which the local effects of the tartar-emetic, vomiting and purging, are not produced, is very small. Grisolle noticed only twelve cases in 154 patients. This want of local action must be regarded as an unfavorable symptom, for of these twelve patients nine died. Vomiting usually occurs within an hour after the first dose, and this is soon followed by purging. Usually the purging is more frequent than the vomiting, and the latter is the first to cease. Tolerance is usually established by the third day. The number of evacuations vary from six or eight to twenty, or more, in the twenty-four hours. The vomiting is more or less distressing, but the purging is generally free and easy.

It has been thought that an irritable or inflamed condition of the *primæ viæ* was a contra-indication to the tartar-emetic treatment. Undoubtedly, when any decided evidences of inflammatory action exist, this remedy should be avoided. But its efficacy in the treatment of pneumonia is so great, that you must be well convinced that such a condition exists, before you proscribe its use. Grisolle found, in many cases, in which slight

colicky pains with diarrhœa and even abdominal tenderness existed, that the moderate exhibition of the tartar-emetic (six to eight grains) relieved rather than aggravated the abdominal symptoms. In such cases, indeed in all cases where too much local action from the remedy is to be feared, you had better combine a certain quantity of opium with it. M. Grisolle thinks, however, that opium has very little influence in controlling the action of the remedy.

The same excellent observer, in his extensive experiments with the tartar-emetic, found that, in a series of cases in which bleeding alone was resorted to, the patients having, at the time of commencing the treatment, the *first stage* of pneumonia, the duration of the disease was ten days—the mortality one in ten cases; while in those in which the disease had passed into the *second stage* before the treatment was commenced, the duration was twelve days—the mortality one in six cases.

In those treated by tartar-emetic alone, cases in which the disease was in the first and second stages, generally in the second stage, the duration of the disease was ten days—the mortality one in seven cases.

In those cases treated by blood-letting first, and by tartar-emetic afterwards, in the first and second stages of the disease, mostly in the second stage, and in which venesection might still be resorted to with propriety, the duration was fourteen days—the mortality one in eight cases.

The favorable influence of the tartar-emetic treatment in pneumonia, that is, in the primitive pneumonia of adults, is one of the clearest points of therapeutics. Old age offers no exception to its use—feebleness of constitution no insurmountable objection. Like every other powerful remedy, its administration must be regulated by the strength of the constitution, the susceptibility to its influence. Care, also, must be taken that it be not used when a decided inflammatory action exists in the primæ viæ; but you need not allow a trifling irritation there to interfere with its use, as such symptoms are often removed, instead of being aggravated by its employment.

In ordinary cases of pneumonia, then, it is proper to bleed pa-

tients once or twice, and to the amount of sixteen or twenty ounces at each blood-letting, if the pulse be full or resistant; but if it be soft, and rather feeble, and the constitutional reaction is moderate, it will not be necessary to practise venesection at all. If there is much pain in the side, a free cupping should be practised. The use of the tartar-emetic, when the pulse is in a proper condition, may be commenced. In well-marked cases, in which the disease is active, and the constitution is vigorous, you may dissolve eight grains in four ounces of water, and administer a table-spoonful every two hours, until the whole quantity has been taken. The succeeding day, the same dose may be repeated, or increased or diminished, according to the state of the patient. If the disease is progressing, it may be increased; the dose may even be doubled in urgent cases. If, on the other hand, the symptoms have decidedly improved, the same dose may be continued, or even diminished. In feeble constitutions, in which the reaction is moderate, and the disease limited, four grains, or even two grains, in eight ounces of water will be sufficient, a table-spoonful of the solution being administered every two hours, as has already been stated.

In advanced cases of the disease, even when there has been reason to fear that suppuration was commencing, and when venesection has been quite out of the question, the tartar-emetic has been administered even in full doses, and with the best success. Local depletion is also useful in many cases of pneumonia in which general depletion cannot be used. It is most marked in its effects in removing the pain in the affected side. Cupping is generally better than leeching. Blisters are stimulants; they are chiefly useful when the disease is stationary, and resolution is slow in taking place. They are also useful in cases in which the pain in the side continues obstinate after the other symptoms are removed, or have much abated.

There are cases of pneumonia, especially those which have been treated too actively at the commencement, or in which the reactive power is feeble from bad habits or a delicate constitution, in which the disease, having reached its full development, remains stationary, or resolves itself very slowly. In these cases a complete change of treatment is indicated; you may prescribe

blisters, stimulating expectorants, and even wine. Some practitioners would use mercury in such cases.

In ordinary cases, the disease advances rapidly to convalescence: as the diet is more nutritious, the strength improves without any other assistance from art.

Typhoid pneumonia does not require blood-letting, as a general rule. In some epidemics, a free blood-letting at the very onset of the disease, has been productive of advantage. Local blood-letting may often be employed with benefit, but according to the circumstances of the case. A supporting treatment—wine, and the stimulating expectorants are indicated.

Mercury has long been used as a remedy in the treatment of pneumonia—calomel in particular. It is undoubtedly a remedy of great power. Statistical comparisons are, however, wanting to establish its relative value in cases of pneumonia, especially as compared with the tartar-emetic. My own impression is, that it is decidedly inferior to that remedy. But it may be used with advantage in cases in which the tartar-emetic cannot be well employed. In cases in which pneumonia exists with dysentery, or with enteritis, it may be used in combination with opium.

Opium is indicated in cases in which the cough is very troublesome, and when there is no sleep. Dover's powder is the best form of administration; or, if combined with antimony, morphine. In the delirium of drunkards affected with pneumonia, it may be used freely, and is of great value. It has been thought to diminish expectoration, but this effect is doubtful.

The diet in the acute stage of pneumonia should be exceedingly simple—gruel, arrow-root, toast-water. When the violence of the disease is passed, and the appetite begins to return, a more free allowance of food may be permitted: first, farinaceous food, then broths, and finally solid animal food.

There are cases of even simple pneumonia which require the moderate use of animal food during the whole progress of the disease—not solid food, but light broths.

There are other cases, still more numerous, especially at the advanced period of the disease, which may require even wine, and the most active supporting regimen.

Drunkards are generally better for a little stimulus during the

disease. It does not aggravate the pneumonic symptoms, and tends to relieve those of delirium tremens, if they occur.

The warm bath has seldom been used in the pneumonia of adults. Chomel has used it with advantage in cases in which the disease was advanced in its progress, and the skin continued hot and dry, the other febrile symptoms having become ameliorated. Great care should be taken in its administration, that the patient is not chilled in leaving the bath.

In the bilious form of pneumonia, the first point in the treatment is to evacuate the alimentary canal by an emeto-cathartic. In cases in which the pulse is full and strong, it may be necessary to precede this treatment by venesection. But you must be careful how you bleed patients in this form of the disease, as it is apt to assume in its progress a typhoid character. After the bilious element of the disease has been removed by one or more emeto-cathartics, the case enters into the category of simple pneumonia, and becomes, usually, a mild form of disease.

When pneumonia occurs during the progress of an intermittent fever, local and sometimes general blood-letting are necessary to mitigate the inflammatory symptoms. But you must remember, in these cases, that quinine is the chief remedy, and not allow the existence of a local inflammation to deter you from the use of anti-periodic remedies. For the first indication is to neutralize the miasmatic influence, and when that is accomplished, to combat the local inflammation, if it has not already yielded.

In secondary pneumonia, the local affection is subservient to the primary disease, and the treatment must be regulated in reference to it. As a general rule, pneumonia occurring in the course of a febrile affection, as continued fever, does not bear general depletion as well as the primitive form of the disease, but much depends upon the stage of the primary affection in which the disease of the lung is developed. The general character of the primary disease must be attentively considered. As a general rule, the earlier in the disease the pneumonia is developed, the better will bleeding be tolerated. The second consideration is of equal, or even of greater importance. Thus, venesection will be better tolerated in a pneumonia secondary to acute rheumatism than secondary to typhoid fever. In this

latter affection, indeed, in most cases of secondary pneumonia, local depletion is much to be preferred to general bleeding. You may ask the question, whether in secondary pneumonia you shall use the tartar-emetic? By all means, unless there is some decided contra-indication, a gastro-intestinal inflammation, or a typhoid tendency, or great debility.

In the primitive pneumonia of children, the same indications of treatment exist as in the primitive form in the adult: venesection, or local depletion, according to the age and condition of the patient, and tartar-emetic, are the principal remedies. To the younger class of children (two to five years of age) you may apply from four to six leeches; to older children (six to fifteen years of age) you may apply from eight to fifteen leeches. In those who are younger, you had better confine your depletion to leeching. It acts in them both as a means of general and of local depletion. The tartar-emetic may be used in doses of two, four, or six grains in the twenty-four hours, according to the age of the child, and in divided doses of one-eighth to one-fourth of a grain. In many cases, in which it is doubtful whether the antimony will be well tolerated, ipecac is an excellent substitute. Many practitioners prefer it to antimony, except in very urgent cases.

Warm bathing, with proper precautions, is a very valuable aid to leeching and to the tartar-emetic, in the pneumonia of young children. It may be used at the same time with these means, and the child may be kept in the bath until some degree of faintness is induced.

If convulsions occur, or other symptoms of cerebral disease, the condition of the lungs must still claim your attention. Leeches, however, will often be of service when the head is hot and the face flushed. They may be applied to the temples or behind the ears. Cold applications to the head are also indicated, and the child must be protected from the light, and be kept as quiet as possible. After the febrile excitement has been subdued, blisters to the head, or to the neck, or to the lower extremities, may be applied with advantage. Blisters, however, should always be used in young children with great caution. If they act too violently, or are used too early in the

treatment, they are apt to aggravate the fever and the restlessness.

Secondary pneumonia, when it occurs in young children, and this you will remember is the most common form of the disease, is not usually developed as rapidly as the primitive form. When it succeeds to an attack of capillary bronchitis, as is usually the case, and the child has previously been robust and healthy, it is an acute disease requiring prompt and active treatment. As the disease attacks both lungs, it is the more formidable on this account. When, therefore, you suspect that the bronchitis is passing into the more grave form of disease, you must not wait until all doubts are removed by the signs of a well-developed pneumonia—until the dulness on percussion and the bronchial respiration have overcome the subcrepitant rattle, and taken its place; but you must resort to active depletion—active, according to the vigor of the patient, the constitutional reaction, and the degree of oppression in the chest. You must practise venesection, if the child is old enough to admit of it, apply leeches to the posterior portion of the chest, use the tartar-emetic in frequently repeated doses, unless some other complication forbids its use, administer the warm bath, and keep the patient on low diet, and as quiet as possible. All cases, however, do not admit of such active measures. A more feeble constitution and a moderate degree of febrile reaction, will perhaps be most benefited by the moderate application of leeches to the posterior parts of the chest, followed by the use of ipecac in nauseating doses. The Syrup of Ipecac, combined with the Spirit of Mindererus, is a good prescription in these cases, which often require much delicate management, especially in avoiding too great a reduction of the vital powers. I think it may be stated as a general rule, that the secondary pneumonia of children in an acute form, like the secondary disease in the adult, requires less depletion than the acute primitive form of the disease, and that it is more under the influence of local remedies, and especially of local depletion.

When the secondary pneumonia of young children follows other acute diseases besides bronchitis, it is particularly modified by the character of the disease. In continued fever, in the

eruptive fevers, it often assumes an active and violent form, requiring prompt and active measures of relief. There is, however, a great difference in these diseases. In continued fever, especially the typhoid form, in scarlatina, much less active depletion is usually necessary than in measles or in small-pox. Indeed, the asthenic character of the original disease may only be aggravated, increased, by the supervention of the pneumonia. Although local depletion, used with the greatest caution, may sometimes be necessary, yet you may find it important to combine it with internal stimulants, thus keeping up the strength, while you attempt to remove the local disease.

Finally, when secondary pneumonia occurs in the course of some chronic disease, and in children of very feeble stamina, a supporting treatment can alone be thought of; but I may add, with very little prospect of advantage. The stimulating expectorants, a nutritious but simple diet, the warm sulphur bath may be used; but, generally, the disease slowly progresses, with very little excitement and very little cough, but with more or less marked acceleration of the respiration, until at length the fatal issue arrives.

LECTURE IX.

PLEURISY.

Effusion of serum; lymph; pus; blood.—Compression of the lung.—Primary and secondary pleurisy.—Acute and chronic pleurisy.—Causes.—Duration.—Mortality.—Symptoms of primitive acute pleurisy; of subacute pleurisy; of secondary pleurisy.

When you open the chest of an individual who has died of *primitive acute pleurisy*, you will find the pleural cavity of the affected side more or less filled by a fluid secretion, and the surface of the pleura covered by layers of lymph. These effusions compress the lung, reducing its size and forcing it inwards and backwards towards the spine. If you examine the effused

fluid, you will find it yellowish and turbid in appearance, being, in fact, composed of serum, in which numerous minute flocculi of lymph are suspended. If a portion of the effusion be placed in a glass vessel and allowed to stand for a short time, it separates into its two component parts. The clear, straw-colored serum floats above, while the lymph subsides, as a sediment, to the bottom of the vessel. The quantity of liquid effusion varies very much in different cases, but, generally, from one to three or four pints will be found. After removing the fluid contained in the cavity of the pleura, you will find the surface of the pleural membrane covered everywhere by layers of lymph—yellowish, opake, friable, easily separated from the pleura beneath. Sometimes the lymph presents itself as a thick, irregular, spongy mass, especially along the free edges of the lobes of the lung, while in other portions it is more solid and more uniform, and easily separated into layers. If, after removing the effused lymph, you examine the pleura itself, you may be surprised to find it hardly altered in any of its physical properties: it retains nearly its natural transparency and delicacy. In most cases, however, you will find it somewhat tarnished, and a little thickened. Sometimes, indeed, these changes are quite decided. If, again, you look beneath the pleural membrane, you will notice that the sub-serous cellular tissue is more or less injected by minute red points, and by short, tortuous lines. Indeed, as in other forms of serous inflammation, the chief seat of the inflammatory action is in the cellular membrane beneath the serous membrane, while the consequences of that inflammation are exhibited upon the free surface of the pleura.

The lung itself, in simple acute pleurisy, is seldom affected by the surrounding inflammation. At the onset of the disease, it is compressed by the copious effusion, and thus, with its natural functions arrested, deprived in a great measure of its blood and air, it remains unaffected in its structure. When examined, it appears wrinkled, flaccid, and without crepitation. Its vessels contain but little blood. You will easily understand why a lung thus compressed and inactive should resist the progress of the inflammation that surrounds it. But independently of this, the tendency of serous inflammations, generally, to affect

the subjacent tissues is but slight, as is proved by studying the progress of the inflammation in other organs, where compression plays no important part.

In cases in which the disease has passed its acute stage, and terminated fatally after several weeks' duration, or even after having continued for several months—in simple chronic pleurisy, you will find that the liquid effusion has become purulent, that the lymph covering the pleura, as well as the pleura itself, is more thick and opake, and that the lung, atrophied perhaps by the long-continued pressure, is still more reduced in size. Indeed, I have seen the lung, when removed from its position against the spine, no larger than the first two fingers of my hand; and in this case, it was evidently not only diminished in size by being deprived of its air and blood, but actually atrophied by long-continued inaction and pressure. It was such cases, no doubt, which led the ancient pathologists to believe that the lung was sometimes destroyed entirely by suppuration. The wasted lung, buried beneath thick layers of lymph, escaped their observation.

The formation of pus, in cases of primitive pleurisy, seems to be gradual. It is only in advanced cases that it assumes the characters of pure pus. In the cases which terminate fatally after a few weeks' duration, the effusion is thin, and is evidently composed, in part, of serum. It has, very properly, received the name of sero-purulent effusion. Probably, also, in an earlier stage of the disease, when it is still in its acute stage, a certain number of pus globules exist in the effused serum.

Sometimes, especially in persons of feeble constitution, you will notice cases, which, if measured by the time the disease had existed, would be called cases of chronic pleurisy, but in which, after death, an abundant serous effusion and but very little lymph or pus exist. These cases, as I shall have occasion to remark again, seem to be developed by the existence of a low degree of inflammatory action, which does not advance much beyond the effusion of serum, but which, occurring in feeble constitutions, and developed insidiously, is sometimes protracted to a fatal termination.

Again, while the effusion in pleurisy may be protracted with-

out any marked tendency to the formation of pus, so, on the other hand, when the inflammation is violent in its character, pus may be early secreted, and the patient dying in the early, or acute stage of the disease, the effusion is found to be quite purulent in its character. So that the existence of well-formed pus in the cavity of the pleura cannot, justly, be considered as indicating the stage of the disease in every instance, although this is true as a general rule. The history of the case, during life, must always be carefully studied, and then you will observe that, generally, where well-formed pus is found, the disease has lasted for several weeks, perhaps for several months, while, occasionally, the disease has terminated fatally after a few days' illness. In other cases, still more rare I think, an abundant effusion of serum, mixed with a few flakes of lymph, are the chief traces of a protracted disease.

I have thus far spoken only of the appearances presented in fatal cases of simple or primitive pleurisy. Fortunately, these are the exceptional cases. You will have abundant opportunities of examining the appearances presented after a cure has been effected, the patient dying of some other disease; for pleurisy is one of the diseases in which the parts affected are very seldom, if ever, completely restored to their natural condition. The serum is absorbed, but the lymph is not, or only partially, and as the opposite surfaces of the pleura are brought in contact again, by the removal of the pressure and by the consequent expansion of the lung, the two surfaces covered still by lymph adhere, the lymph becomes organized, and the cavity of the pleura is more or less completely obliterated. In cases in which the effects of simple acute pleurisy have been observed, I have found the union of the lung to the ribs more or less complete in every part; sometimes more closely attached, sometimes more loosely, by the intervening organized lymph. The lung itself, in such cases, is more or less deformed, of course, by the lymph that invests it; but more than this, it is usually rather smaller, and less perfectly aerated than is natural.

When chronic pleurisy has terminated favorably after a long struggle, and the patient dying, at a remote period, of some other disease, you will find the lung much more materially af-

fected. The long-continued pressure and inaction have induced a certain degree of atrophy, and the thick false membranes which have covered it have interfered with its free expansion; so that it is far from having regained its natural dimensions, its full development. Still, in the worst cases, the crippled lung does not remain entirely useless: it still, imperfectly indeed, performs its functions; and its imperfection has been, in part at least, compensated by the increased development of the sound lung, which has increased in size, and of course in function.

A careful examination, therefore, of the effects of primitive pleurisy, abundantly proves that the organic changes it produces are seldom, if ever, entirely removed, and that the lung does not recover entirely its former full development and function. Thus there is this striking difference between pleurisy and pneumonia —in the latter disease, the lung rapidly regains its functions, and no traces are left of a formidable disease, except, perhaps, in the trifling adhesions which mark the coexisting pleurisy.

It very seldom happens, when you examine the lungs, particularly in the adult, that you do not find traces of pleurisy in the adhesions which exist in the opposite surfaces of the pleura. These adhesions being usually limited in extent, confined perhaps to the apex of the lung, or to the interlobar spaces, indicate, in many cases, the existence of a very slight pleuritic attack, during which a small quantity of lymph was thrown out, producing the adhesions in question. The perfectly healthy condition of the lung would not allow you, at first sight, to refer these adhesions to any antecedent disease in that organ. Yet, when you remember that primitive pleurisy has a decided tendency to diffuse itself over the whole membrane, and thus produce a general adhesion, you cannot doubt that many of these partial adhesions are owing to some limited disease of the lung itself, a pneumonia, for instance, all traces of which have disappeared. Indeed, it must be admitted, I think, that most partial pleurisies are *secondary*, and dependent upon some antecedent disease in the lung. The deposit of a few tubercles, for instance, in the lung, leads to this result, and this is undoubtedly a frequent cause of the traces of partial pleurisy which are noticed after death. Another circumstance connected with these sec-

ondary pleurisies is worthy of notice: they constitute what have been called dry pleurisies. The inflamed pleura throws out a little lymph only; there is no serous effusion. Even in cases of pneumonia, in which a most active inflammation exists, the effusion into the cavity of the pleura is, usually, limited to a little lymph, which is sometimes so thin and transparent, as to readily escape observation.

There is a variety of pleurisy sometimes met with, and which has been regarded as a secondary affection—the *hemorrhagic pleurisy*. In this variety, a certain number of blood globules may exist in the serous effusion from a rupture of the capillaries, giving it and the effused lymph a more or less deep red color; or a reddish or greenish yellow tint may be imparted to the fluid by the simple effusion of the coloring matter of the blood. In these cases the serous effusion is usually abundant, while the quantity of lymph noticed is moderate in amount. The hemorrhagic tendency has been supposed to be connected with the existence of tubercles in the lungs; but I have known it to exist when the lungs were entirely free from the tubercular deposit. I doubt, however, if it ever occurs in those who are free from previous disease, especially disease of a chronic character, by which the powers of the constitution have been much exhausted.

Finally, you will meet with cases, but they are very rare, I think, in which a pleurisy occurs which is limited by the existence of previous adhesions, and which seem to resist the progress of the recent inflammatory action. In these cases, a limited pleurisy may terminate in the formation of pus; in fact, in the formation of what might easily be mistaken for an abscess in the lung. The pus thus secreted, circumscribed by old adhesions, presses upon and indents the subjacent portion of that organ, so as to produce an apparent loss of substance. But, after a careful examination, it will be evident that the collection of matter is entirely external to the lung in the cavity of the pleura.

The tendency of all the varieties of pleurisy, except, perhaps, the hemorrhagic and the essentially chronic variety, to terminate in adhesion of the opposite surfaces of the pleura, is a well-established fact. Few cases, I believe, even under the best treatment, escape from this result. These adhesions becoming organ-

ized, are gradually converted into delicate and transparent membranes, which seem to give little or no subsequent trouble. But, in certain cases, the organic changes do not stop here. If pus exists for a long time in the pleural cavity, even if it is limited in quantity, and, still more, if tuberculous disease is progressing in the lung, these membranes become gradually more thick and opake, and more firm in their structure. In some cases, before evidences of their organization are noticed, they can be peeled off from the surface of the pleura in large and thick strips, looking like portions of wet buckskin. When, however, these membranes have become organized, and continue to be subjected to irritation, they become still more firm and fibrous in their character: they may even assume the appearance of fibro-cartilage, or of bone.

The pleura itself, which lies beneath these effusions of lymph, is usually found much thickened and opake, as also the subserous cellular tissue.

It is supposed that pus, when secreted into the cavity of the pleura, may, by a partial decomposition, generate a gas, and thus that the chest may become the seat of a hydro-pneumothorax—the upper portion of the cavity of the pleura being filled by gas, while the lower portion contains a purulent fluid, the relative proportions of which vary in different cases. I have never met with such a case. Every case that I have examined has evidently been dependent upon the existence of tubercles in the lung, which, by softening and ulceration, have probably established a communication between a bronchus and the cavity of the pleura. The consideration of these cases properly belongs to the subject of tubercle.

Pleurisy is sometimes *double*. It is the opinion of Louis, a very high authority certainly, that in this case the disease is usually associated with tubercles in the lungs, and always with some form of organic disease of the lungs. In this case, double pleurisy is a secondary form of the disease.*

* According to Vogel, the distended capillaries in inflammation having their walls thinned by this distension, exude serum and fibrine at the same time—a fluid which is identical with blood without its corpuscles. After a time this fluid coagulates, and the fibrine is separated, forming a coagulum of lymph, of more or less

You will find that pleurisy occurs chiefly in the latter part of the winter, and during the spring months. If you take the ex-

firmness, which, under the microscope, appears either perfectly amorphous, or exhibits a confused fibrous or radiated appearance, and is sometimes covered by a finely granular or pulverulent matter. When the fluid fibrine has assumed the coagulated condition, and has become perfectly isolated, the remaining fluid, in every respect, corresponds with the serum of the blood.

This fibrinous fluid is capable of organization, and it is indifferent whether the fibrine is in a fluid or in a coagulated state, and from it may be evolved the most different forms of tissue, either normal or pathological. The first indication of the developmental process in the coagulated fibrine, is the development of cells—until then it is amorphous.

This fibrinous exudation is the uniform source of pus. The pus corpuscle may form either while the fibrine remains fluid, or after it has become coagulated. In examining the fluid exudation, minute granules are first observed—then somewhat larger corpuscles, identical with the nuclei of the pus corpuscles. Around these, a cell-wall is subsequently developed, at first pale and transparent, then thickened and granular. Thus the true pus corpuscle is formed (Appendix, Pl. 1, figs. 6 and 7). [Lebert takes a different view of the formation of the pus corpuscle. In the effused liquid are formed little globules, round or with an irregular outline, and containing small granules which are afterwards developed into nuclei. He also describes a variety of the pus globule, pyoid globule (Appendix, Pl. 1, fig. 12, B)]. The formation of the pus globule in the coagulated fibrine is probably the same as in the liquid form. The pus corpuscles, however, are seldom detected until they are perfectly formed. At first they are scantily dispersed through the amorphous or indefinitely fibrous coagulated mass. Subsequently, however, they become more abundant, and occupy the whole mass, being separated from each other by intervening serum—the solid portions of fibrine disappearing, and the whole of that constituent being converted into fluid pus.

Pus, then, is a liquefaction of the solid fibrine. But although fluid, it cannot be absorbed, except the serous portion, until the pus globules are broken up into molecular granules (Appendix, Pl. 1, fig. 1, *b*). This process seldom takes place; therefore pus is seldom absorbed, but exhibits a strong tendency to find an external exit.

Coagulated fibrine is also liquefied by another, but analogous process. Large granular cells (Appendix, Pl. 1, fig. 12, C) form in it. These more readily break down into molecular granules, the mass is liquefied, and is frequently absorbed.

Lebert thus describes the subsequent changes in the fibrine which is not liquefied: All the liquefied portions are absorbed; even the pus globules pass into a state of granular diffluence. The remaining fibrine thus becomes more and more condensed, and the appearance of fibrous stratification is changed by degrees into a true fibrous tissue by condensation. It is thus that the adhesions of the lung to the ribs are formed. This fibrous tissue sometimes becomes so dense and white as to resemble cartilage, but it contains none of the true elements of cartilage. Sometimes it is transformed into a plaster-like, or amorphous mineral matter, arranged in plates, and looking like bone, but without exhibiting, under the microscope, any of the char-

perience of this Hospital as your guide, the disease is much less frequent in its occurrence than pneumonia. It is also much less often fatal in its acute stage. It is reasonable to suppose, that the combination of cold and moisture with sudden changes of temperature, operate as predisposing causes and sometimes directly induce an attack. A very interesting class of cases occurs in persons of the scrofulous diathesis, in which the disease seems to follow rheumatism of the muscles of the chest. In other cases the disease occurs often without apparent cause, in the progress of other diseases—in febrile diseases, as continued fever, in the eruptive fevers; also, in the course of chronic diseases, especially in that form of disease of the kidney attended by albuminous urine and by dropsical symptoms. Among the diseases of the lung itself, pneumonia and tubercular disease are almost always accompanied by pleurisy. Indeed, the causes which induce pleurisy must be numerous as well as general in their operation, when it is remembered how almost universally the traces of it are found after death.

The *mortality* in primitive pleurisy is not great.* Most of

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acteristics of the bone structure. Another change, is the conversion of the effused fibrine into a gelatinous matter, yellow, semi-transparent, composed of a hyaline substance, fine fibres, and granular globules, and containing irregular nuclei, appearing like cartilage or bone, but exhibiting under the microscope a dense, fibrous tissue, or an amorphous mineral substance. This is the colloid tissue which sometimes enters also into the composition of cancerous masses. There is, finally, another transformation, by which the inflammatory exudation is changed into bone-like plates, but which contain crystals of cholesterine as their principal element.

* According to the report of the City Inspector of New York, the whole number of deaths from Pleurisy during three successive years was only one hundred and six, while during the same period of time, the deaths from Pneumonia were two thousand five hundred and fifty-eight. Fifty-four of these cases of Pleurisy occurred in males, and fifty-two in females.

The number of cases of Pleurisy which occurred from

December to June, was.....72
June to December,.....34

Proportion of deaths from Pleurisy, to the whole number of deaths in other cities:

Boston, (period of 5 years), 1 in 166 deaths.
Baltimore, " 4 " " 81 "
Charleston, " 5 " " 228 "
London, " 1 " " 603 "

In London, during one year, 75 fatal cases of Pleurisy are reported; from December to June, 51 cases; from June to December, 24 cases.

the cases recover, especially if treated properly in the early stage. The mortality is, indeed, so trifling, that you may with great confidence predict a recovery, if the patient possesses a good constitution, if the onset of the disease is comparatively mild, and the effusion moderate in amount, producing a dulness on percussion that does not extend over more than one-half, or even two-thirds of the affected side. But in cases in which the disease is more violent, or the effusion more considerable, in which the whole side becomes dull on percussion, and is even sensibly dilated, then the chances of recovery are greatly diminished. Such cases, occurring, perhaps, from neglect of early treatment, tend to pass into the chronic stage with the formation of pus. Still they often recover; I suppose they do so in at least one-half of the cases, although not often without leaving more or less serious consequences—a contracted side, dyspnœa and cough.

It is difficult to estimate the *duration* of this disease. I may say, that it continues longer than pneumonia occurring under similar circumstances. In the latter disease, the mean duration is about a fortnight, while in primary acute pleurisy twice that length of time would not exceed the average duration. When the disease passes into the chronic stage, and the patient dies, this event seldom occurs before the end of the second month, or at a later period than the end of the fourth month. When, on the other hand, a chronic case terminates favorably, it is often many months, sometimes it is a year or more, before the case can be regarded as fairly cured, and that too without taking into the account certain consequences of the attack, as pain, and a sense of weakness in the affected side, which may remain for an indefinite period. But while you may look upon pleurisy, both in its acute and in its chronic form, as a protracted disease, you must still remember that it may be rapidly fatal. A violent attack may destroy life in a few days, but these cases are comparatively rare.

A healthy and vigorous young man, after working all day, perhaps exposed to cold and dampness, returns home at night feeling hardly as well as usual. He eats his supper with less than ordinary appetite. He complains of a general discomfort, of a tired sensation in his limbs, and he is disposed to sit listlessly

near the fire. By-and-by he experiences a distinct chill, and, very likely, at the suggestion of some friend, he drinks *something hot*, and goes to bed. During the night he is restless, his skin becomes hot, and he begins to cough and to complain of pain in the chest beneath the nipple. The pain increases, the cough aggravates it, a full inspiration aggravates it, and when morning comes he is too ill to rise, and determines to send for a physician. You will find him in bed, and lying upon his back; his face is flushed, his skin hot, his respiration accelerated; but the chief complaint he makes is of an acute pain, felt below the nipple, which is like a stab when he attempts to breathe fully, and is much aggravated by a dry and harassing cough. His pulse is accelerated, probably more than one hundred in a minute, and firm and hard. His tongue is covered by a white coat, he is thirsty, he has no appetite, but if he has attempted to eat any thing he has probably vomited it. The secretion of urine is scanty, and no evacuation from the bowels has taken place since the attack commenced.

If you examine the chest at this early period of the disease, you may have arrived before the earliest, that is to say, the serous effusion has occurred. In this case, no physical signs may be apparent, unless, indeed, a slight feebleness of respiration over the chest, especially over the side where the pain exists, and which is simply owing to an inability to expand the lung from the pain the effort induces. But if, as is more likely to happen, serous effusion has already taken place, and in considerable quantity, you will notice the physical signs of its existence. The lung is compressed by it, or floats upward on the surface of the fluid, so that you will find the lower and the lateral portions of the side of the chest affected, dull on percussion, the respiratory murmur absent, or very indistinct; and if you listen to the vocal resonance, you will often catch over the seat of the dulness, the silvery, vibrating note of egophony.

Such is the history of the development of a case of primitive pleurisy in the adult.

You should prescribe a free venesection, the patient sitting upright in bed until fainting is threatened; you will repeat it if the reaction is considerable; you apply leeches or cups to the

affected side, if the pain continues to be troublesome, and administer the tartar-emetic in full doses.

With this treatment, continued with an absolute diet and rest, the febrile excitement subsides, the pulse becomes more soft and less frequent, the pain in the chest is removed, or is hardly noticed, and the dyspnœa abates. But with this general improvement in the constitutional and rational symptoms, the physical signs continue, and are probably more extended than at the commencement of the attack. The dulness extends, perhaps, as high as the second or third rib in front, the respiratory murmur is heard, indeed, at the summit of the lung, and between the scapula and the spine, the lung being pressed backward in this direction, but elsewhere there is no sound. The egophony has also disappeared, and given place to a feeble resonance of the voice. Gradually, however, and generally without the use of special remedies, the liquid effusion is absorbed, the lung expands again, the dulness diminishes, and the respiratory murmur returns. These physical changes proceed, of course, from above downward, but even after the recovery may be considered sufficiently advanced to render a longer attendance unnecessary, a pretty decided dulness on percussion and feebleness of the respiratory murmur remain over the lower portion of the lung, and will continue to exist for a long period, the compressed lung not regaining its full expansion in its lower lobe.

In this sketch of a case of primitive acute pleurisy, I have only alluded to the early treatment; I shall speak more fully on this subject on another occasion. I will only add, that after the active inflammatory symptoms have been reduced by the antiphlogistic remedies already mentioned, it will often be proper to use mercury in alterative doses, and afterwards to administer diuretic remedies, as means of promoting the absorption of the lymph, and of the effused serum.

If the attack is more severe in its character, or is neglected in its early stage, the effusion in the chest becomes more copious, more rapid, and in a few days you may find it so considerable as to fill the cavity of the pleura, and even to distend the parietes of the chest, and to displace the heart or liver. Every portion of the chest over the affected side is very dull to percussion,

no respiratory murmur is heard, except between the scapula and the spine, the resonance of the voice is very feeble. But with these aggravated physical signs, the pain in the chest may be but trifling, and the febrile excitement moderate, yet the oppression of the respiration is great, the pulse much accelerated, and the vital powers are giving way. A patient thus affected may sink and die during the acute stage, or, what is more common, the disease may remain nearly stationary for a time, and, finally, become chronic. If pus predominates in the effusion, as it usually does when the disease has continued for several weeks, hectic symptoms appear, and the general condition of the patient deteriorates more rapidly than before. He may die from exhaustion, or, under appropriate treatment, the effusion may be absorbed, or it may discharge itself externally between the ribs, or in certain rare cases, by communicating with a bronchus. Under all these circumstances, however, the recovery is slow. The side affected gradually becomes contracted, and the lung regains only in part its functions.

An attack of acute pleurisy may be so violent as to pass rapidly to the formation of pus even without much serum or lymph being effused. The disease passes rapidly by the stage, when serum and lymph are alone effused, and which indicates a milder degree of inflammation. In such a case, the disease may rapidly pass to a fatal issue, with the most active constitutional and rational symptoms, and yet the physical signs be by no means prominent. A moderate extent of dulness, indicating a moderate compression of the lung, and yet violent symptoms and a fatal issue—facts easily explained when you examine the cavity of the pleura after death, and find the serous membrane highly inflamed, and bathed in pus.

The *primitive pleurisy of young children* does not possess any striking anatomical characteristics which distinguish it from the disease in adults, as is the case in pneumonia. There is, however, one fact in these cases which distinguishes the affection: it is the comparative want of compressibility in the lung from the liquid effusion; and the consequence of this is perceived in the modification of the physical signs. The dulness on percussion presents its usual characteristics as to extent

and degree. But the respiratory murmur, instead of being feeble or absent, assumes a *bronchial character*, which is equally distinct as that of pneumonia, but far more extensive, accompanying the dulness on percussion, and being often heard all over the affected side, and without crepitation or rhonchus. The constitutional and the rational symptoms do not differ from what is noticed in the acute pleurisy of adults, allowance being made for the greater tendency to nervous excitement which exists in children, and of course the greater probability of the existence of cerebral symptoms, as convulsions, delirium, or coma.

There is a form of pleurisy which may be called *subacute*, and which possesses great interest from its connection with the tuberculous diathesis. This form of disease, so far from being severe at its onset, is remarkably the reverse. It commences with wandering pains about the chest, like those of muscular rheumatism, and is, at first, unattended by cough, or by dyspnœa. Usually there is no perceptible febrile excitement, although this is sometimes noticeable. After this condition has continued for several days, the patient begins to complain of dyspnœa, which rapidly increases, and on examining the chest, the physician is surprised to find the evidences of a considerable effusion into the pleura. At the time the effusion occurs, the inflammatory symptoms become more decided, the pulse is accelerated, the skin warm, and cough supervenes. But these symptoms are mild in their character. These cases are serious, not in their immediate, but in their remote consequences. I have observed in many such cases, that the pleuritic symptoms were followed by those of the tuberculous deposit, which more or less rapidly proved fatal. A patient thus affected will appear to be recovering from his pleuritic attack—the febrile symptoms have subsided, the effusion is being absorbed. But this favorable progress is often interrupted. The pulse becomes accelerated, night-sweats supervene, and the strength does not improve. No further improvement takes place in the physical signs. The dulness on percussion does not diminish. A bronchial respiration, with mucous rattle, supervenes over the seat of the dulness, or at the summit of the lung. The opinion was ex-

pressed many years ago by Dr. Gerhard of Philadelphia, that pleurisy might terminate in tuberculous disease. It is in cases like that I have just described, that I have noticed this result. It may happen, indeed, that latent tubercles have existed in the lung before the pleuritic attack, and that the inflammatory disease has simply called them into activity. This is a reasonable view of the matter, and some cases that I have seen would favor this opinion, the patients having at some previous period of their lives been affected by at least suspicious symptoms of tubercles. But in the largest proportion of cases no such previous symptoms have existed. Those affected, have been delicate persons of the scrofulous diathesis, having lost, perhaps, near relatives by tuberculous disease, yet themselves previously unaffected by any pulmonary symptoms. My own observation has led me to regard this insidious subacute form of pleurisy, which commences like an attack of muscular rheumatism, as the most serious form of the disease in its ultimate prognosis. I am happy, however, to state that an unfavorable result is not always realized. Some cases recover perfectly.

The development of this form of pleurisy is so insidious, that I will occupy your time by relating a few cases.

A gentleman, about thirty years of age, of a delicate constitution, but in the habitual enjoyment of good health, having lost near relatives by tuberculous disease, was first attacked with pain in the back, which was regarded by his physician as lumbago, especially as no pulmonary symptoms were present, and there was no febrile excitement. After a few days, the patient began to be affected by dyspnœa, especially in going up stairs. This led to an examination of the chest, and a copious effusion was found to exist in the left pleural cavity. The mode in which this case developed itself, as well as the constitution of the patient, led me when called in consultation, to form an unfavorable prognosis, although nothing but the evidences of pleurisy as yet existed; and this opinion was justified by the result. The case improved for a time; the dulness on percussion diminished, but finally remained stationary, with a feeble respiration and an occasional mucous rattle over the part affected. After an interval of a year, this case terminated fatally as tu-

berculous phthisis—a fact confirmed by post-mortem examination.

A gentleman, also about thirty years of age, whose mother had died of phthisis, and who had himself exhibited some suspicious symptoms several years before, met me one morning in the street, complaining of pain in the left side. This pain was shifting in its character like rheumatic pain, and accompanied by a good deal of external tenderness: there was no dyspnœa, no cough, no physical signs of effusion in the chest. Still, there had been some chilliness, and the pulse was accelerated, but the patient was a man of a very nervous temperament, and easily excited. The patient was kept in bed, and simple means, especially counter-irritation, were used to remove the pain in the chest. But the pulse continuing to be accelerated, and some heat of skin being present, he was bled moderately as a precautionary measure. The pain in the chest subsided, the febrile symptoms ceased, and the patient seemed fully convalescent. Nearly a week had now elapsed since the attack, when he was suddenly seized with great dyspnœa, and on examining the chest, a copious effusion into the left pleural cavity was discovered. Cough now supervened for the first time, the febrile symptoms reappeared, and the patient was again confined to his bed. But in due time the pleuritic effusion began to be absorbed, the dulness on percussion was diminishing rapidly, when, without apparent cause, these signs of improvement ceased. The dulness on percussion continued unchanged, a mucous rattle supervened in the same region, and afterwards in front, towards the summit of the lung, the pulse became much accelerated, and copious night-sweats ensued. In a word, the symptoms of tuberculous disease began to appear.

Secondary pleurisy, when it occurs in the course of acute or chronic disease, like pneumonia, is modified by the symptoms of the original affection. The statements that I made to you in a preceding lecture, in relation to secondary pneumonia, apply exactly to secondary pleurisy. In acute and inflammatory diseases, the occurrence of pleurisy aggravates the general condition of the patient, but is seldom attended by any strongly

marked rational symptoms, unless it be by dyspnœa. The physical signs, however, reveal the true nature of the complication. In chronic disease, pleurisy may assume a latent form, the physical signs even may be indistinct, and after death a small secretion of pus may be found in the pleural sac. In other cases, however, a much more abundant effusion of fluid may occur, the parietes of the chest may even be dilated by it, and after death a hemorrhagic pleurisy may be detected.

The occurrence of a *double pleurisy* with liquid effusion is, I think, a more rare disease than a double pneumonia, even in adult life. When it does exist, it is, according to the testimony of Louis, a secondary form of the disease, and often connected with the existence of tubercles, although sometimes with other organic affections of the lungs. In such cases, the dyspnœa and distress of the patient is much aggravated, and the constitutional symptoms assume a grave character. In this form of the disease the prognosis and the treatment are equally unfavorable.

LECTURE X.

PLEURISY.

Chronic pleurisy.—Symptoms and physical signs.—Diagnosis of pleurisy from pneumonia and from bronchitis.—Treatment of acute pleurisy; of chronic pleurisy.

The physician of a Hospital is frequently called to examine patients affected with pleurisy who have been ill several weeks, or even months. In a large proportion of these cases, it is easy to trace the origin of the disease to an acute attack, occurring suddenly with pain in the chest, dyspnœa, cough, fever, and prostration of strength. Sometimes, however, these early symptoms are less marked, or if they have occurred their existence has been forgotten. A case of chronic pleurisy presenting itself, for the first time, under these circumstances, possesses certain peculiarities which are deserving of notice, as distinguishing the

disease from the acute form. The patient will be found in bed, the countenance pale, and with a look of exhaustion. He is feeble and emaciated, his digestive functions are imperfect, his sleep disturbed. He has no longer the continued excitement of acute inflammation, but he complains of irregular chills, of a disposition to fever in the evening, and of sweating at night. In fact, his febrile symptoms have assumed the form of hectic. His pulse is much accelerated, and rather feeble. He coughs, with a muco-purulent expectoration; he complains of dyspnœa, especially on motion, and finds that he breathes much more easily when lying on one side. He complains of no pain, or if this symptom exists at all, it is slight. If you examine the chest, you will find the most decided evidences of pleuritic effusion—extensive dulness, absence of respiration, dilatation of the affected side.* But you will often find more than this: the external cellular tissue of the affected side has become œdematous, and this has perhaps extended to the arm. The intercostal spaces, especially in the lateral portion of the chest, are more widely separated than is natural, and perhaps give a distinct sense of fluctuation. The case may have advanced still further: you may notice a circumscribed and fluctuating tumor, painful to the touch, and sometimes pulsating from the impulse of the heart. This pointing of the purulent effusion externally, occurs in different portions of the chest. But I have most frequently noticed it anteriorly, between the second and third ribs, where the walls of the chest are naturally thin. Sometimes, however, the pointing is in the lower portion of the chest, and in one instance, it occurred at some distance below the ribs, in the abdominal region.

But every case of chronic pleurisy does not present these striking physical signs. The affected side is not always dilated, neither are the intercostal spaces necessarily widened. Indeed, I believe that cases of purulent effusion sometimes exist, in which the affected side may even appear contracted, probably from the fact that the effusion has been partially absorbed. Of course, in such cases, the dulness, and the absence of the respiratory mur-

* Heyfelder, a German observer, found the left side affected with chronic pleurisy 15 times in 20 cases. Nineteen of these cases occurred in the male sex.

mur, may not affect the upper portion of the chest, and the case, in other respects, presents a less severe aspect than is usual.

The tendency of pus in the pleural cavity to point, and to discharge itself externally, is not the only natural mode of relief. It may happen that an abscess forms in the lung, which, communicating with the pleura and with a bronchus, thus establishes a variety of hydro-pneumothorax. Sometimes, the first suspicion of an abscess in the lung is derived from the sudden expectoration of a large quantity of purulent matter. In other cases, the existence of an abscess may be suspected from the development of a circumscribed mucous rattle in the lung, and afterwards rendered certain by a cavernous respiration. This abscess, indeed, may never communicate with the pleura. Death may ensue, or external pointing take place; and if the patient recovers, the abscess will gradually heal.

Cases are recorded in which the purulent effusion has escaped into the abdominal organs, but such cases are very rare.

It has been said that the effused pus might be removed by a vicarious discharge from some other organ. Thus, it has been supposed that the bronchi might absorb it, and remove it from the system by expectoration. There is no proof, to my knowledge, that this can happen. Probably, in the cases in which it has been supposed to occur, there was a communicating abscess in the lung, such as I have just now alluded to. It is true, that in many cases of chronic pleurisy, there is a more or less copious muco-purulent expectoration. Sometimes, indeed, this is quite abundant. But this is, by a little attention, easily distinguished from the true pus which is secreted in the cavity of the pleura. Again, it has been stated by a high German authority, that the purulent effusion may be carried off by the kidneys, and that a purulent condition of the urine exists in this connection. The proofs of such a statement are wanting to establish a fact so contrary to common observation.

Cases of chronic pleurisy which recover, either by the absorption of the effused pus or by external evacuation, are generally slow in their progress. In cases in which an external opening through the parietes of the chest has taken place, the discharge

of purulent matter often continues for a long time; I have known it to continue for years. This is owing to the fact that the lung, long compressed and perhaps atrophied, cannot expand sufficiently to fill the cavity of the chest, notwithstanding the aid afforded by the contraction of the parietes, which is often very considerable. So long as this condition exists, the cavity will secrete pus, and I know no limit to its duration, although I believe that nature is eventually successful in effecting her object in curing the patient.

Contraction of the affected side of the chest is a permanent deformity in these cases, and often to a considerable degree. The whole side is drawn downward and inward from the top of the shoulder to the false ribs. The spine becomes curved, until at length the deformity can be recognized as the individual walks before you in the street. With this external condition of the chest, the lung never regains its perfect function. There is always dulness on percussion and feebleness of respiration, especially over the inferior portion of the side affected; and, for a long time, an occasional sense of weakness and uneasiness in the side, with dyspnœa and a tendency to cough, continue to exist.

An essentially chronic pleurisy has been described in the books. In this form of the disease, the symptoms are insidiously developed, and consist chiefly of dyspnœa with the physical signs of effusion, without marked pain, or cough, or febrile reaction. Such cases have been described as attended by a copious effusion of serum, and with but little effusion of lymph. I regard them as belonging to the subacute variety of pleurisy, which I have described in my last lecture as occurring chiefly in scrofulous persons, and as often connected with tubercles in the lungs. I do not think that, in such cases, there is much tendency to the formation of pus; so that when the case assumes a chronic form there is but little tendency to pointing, and even the hectic symptoms which develop themselves are more likely to be dependent upon the existence of tubercular disease than upon any other cause.

There is nothing in the constitutional or rational symptoms to distinguish acute pleurisy from pneumonia, in many cases.

The febrile symptoms, in pleurisy, may be more intense, and the pulse, more frequently, hard and resistant. But this is far from being always the case. So with the rational symptoms. The character of the cough, the dyspnœa, the pain in the chest, are the same in both diseases. But from the expectoration in the early stage of pneumonia, you may often derive the most valuable assistance in the diagnosis. The rusty, viscid expectoration of pneumonia is characteristic. But in some cases it is entirely wanting, and, as a general rule, it is only found in the early stage. A case of pneumonia, when compared with a case of pleurisy, after the first eight days from the commencement of the two attacks, would, I think, offer no characteristic differences in any of the rational symptoms.

You are compelled, then, to look to the physical signs, as the chief source of diagnosis in these cases. By this aid, you seldom need be mistaken. The extensive and marked dulness on percussion in pleurisy, often occupying nearly, or quite, the whole extent of the lung, and this united with great feebleness or entire absence of the respiratory murmur, will at once distinguish the disease, in most cases, as one of pleurisy. In pneumonia, the dulness is usually much more limited, and a bronchial respiration, with or without crepitus, exists. If the dulness occupies the upper portion of the chest only, you may know at once that it is not connected with pleurisy, for the effused fluid naturally gravitates to the most dependent portion of the chest. I know that there are a few anomalous cases on record, in which the existence of a circumscribed pleuritic effusion, limited by old adhesions, has produced some doubt in the diagnosis, but such cases are very rare. The existence of egophony, also, in cases of moderate pleuritic effusion—and it is in such cases that the difference in the physical signs in the two diseases I am considering is less marked—will often enable you at once to pronounce a decision.

In cases of pleurisy in which no egophony exists, and this is most likely to happen when the liquid effusion is considerable, the resonance of the voice is usually much less than is natural. The reverse of this is the case in pneumonia, in which bronchophony, or increased vocal resonance, is commonly noticed.

In pleurisy, then, you will notice extensive dulness on percussion, extending into the lateral portion of the affected side, and often reaching to the anterior portion, even as high as the clavicle. With this dulness you will perceive a feeble respiratory murmur, or none at all; and finally, egophony, if the effusion is moderate in amount, or a very feeble vocal resonance when the effusion is more considerable. In pneumonia, you will notice a limited dulness on percussion, sometimes occupying the inferior and posterior portion of the side affected, sometimes the superior and anterior portion, and with this dulness is associated a bronchial respiration, sometimes simple, but often accompanied by a crepitus and by bronchophony.

After stating to you the general rule, I must not forget to notice a remarkable exception. There are certain cases of pleurisy, even cases in which the dulness on percussion extends over the whole side affected, in which a bronchial respiration is heard. This bronchial respiration, however, should be carefully studied. It will generally be noticed that the sound is less superficial than that heard in cases of pneumonia. When the patient is breathing quietly it may not be heard at all. You may hear only a feeble respiratory murmur, or no sound whatever. But if the patient is requested to breathe more forcibly, the bronchial respiration is at once developed, and generally over every portion of the affected side. With these physical signs, you will notice a peculiar resonance of the voice, a compound of egophony and bronchophony, generally known as broncho-egophony. The vocal resonance is clear, vibrating, trumpet-like; or deeper in its tone, like the note of a bassoon, or like that of Punch in the puppet-show.

The reason of these peculiarities in the physical signs of pleurisy is, I think, the existence of any cause which interferes with the compression of the lung. Thus, the existence of miliary tubercles in the lung, or of pneumonia, may produce this result. Another cause is the existence of old and limited pleuritic adhesions, which prevent the lung from being driven away to any considerable distance from the parietes of the chest, when a liquid effusion into the pleural sac occurs. I remember a patient in this Hospital who had dulness on percussion over the whole side of the chest, with bronchial respiration. The side was even

dilated. This case at length terminated fatally, and by post-mortem examination, the lung was found attached to the diaphragm by old adhesions, so that it was prevented from being pushed backward towards the spine, at least to the same degree as in ordinary cases.

This bronchial respiration, as connected with pleurisy, is the rule, not the exception, in the pleurisy of young children. This fact attracted my attention before any writer on the subject, so far as my knowledge extends, had noticed it. I remember very well the first case. It was a child about eighteen months old, who did not appear to be very sick, but had suffered from fever and oppression of the respiration for a few days. I found that one side of the chest was, in every part, dull on percussion, but with a bronchial respiration equally extensive. I could not reconcile these extensive physical signs with the existence of a pneumonia, for with such extensive consolidation, the rational and constitutional symptoms should have been more severe. My attention having been thus drawn to the subject, I was, in time, able to satisfy myself that in the pleurisy of young children the rule is as I have stated it. I can offer no sufficient explanation of this fact. It cannot be explained, as in the occasional cases which occur in the adult, by the pre-existence of a partial consolidation of the lung, or by the pre-existence of partial adhesions. In children the respiratory function is very active, and probably the lung is less compressible than in the adult.

There is a morbid sound sometimes heard in cases of pleurisy, to which I have, as yet, made no particular allusion, because it is rare and usually of short duration. This is a *friction sound;* a dry, rubbing sound accompanying both inspiration and expiration. I have, I think, most frequently noticed it in the lateral portion of the chest, but sometimes anteriorly, near the nipple, sometimes posteriorly, about the lower angle of the scapula. This sound is noticed at the commencement of an attack, before the liquid effusion is considerable, and also for a short time during the absorption of the fluid. It is evidently caused by the rubbing of the false membranes of the pleura pulmonalis and of the pleura costalis upon each other, and ceases when either the liquid effusion becomes considerable, or when

adhesion takes place. I have known, however, this friction sound to continue for a week or longer. It is accompanied, sometimes, by a tremor when the hand is applied to the part affected.

I have now passed in review three acute inflammations of the pulmonary organs—bronchitis, pneumonia, and pleurisy. Let me now compare them with each other in their more prominent and characteristic features.

In bronchitis, the constitutional symptoms are moderate in degree, the cough sonorous, the dyspnœa moderate, the pain trifling. In the milder cases, all physical signs are wanting; but in the more severe forms, especially in secondary bronchitis, in the capillary variety of young children, there is, at first, a sibilant or sonorous rhonchus, and then a mucous rattle developed in both sides of the chest, and especially over the posterior and inferior portions. During the whole progress of the disease the resonance on percussion, as also the vocal resonance, remain perfectly natural.

In pneumonia, the constitutional symptoms are more severe. The cough is suppressed and painful, there is usually a stitch in the side affected, the dyspnœa is considerable, and during the early stage, there is often the characteristic rusty expectoration, but not always. There is limited dulness on percussion, with crepitation and bronchial respiration, usually confined to one lung; there is also bronchophony.

In pleurisy, the constitutional and rational symptoms resemble those of pneumonia, except that the characteristic sputa are wanting. The physical signs, as in pneumonia, are usually confined to one lung. The dulness on percussion is extensive, occupying frequently the whole side affected, and is accompanied by a feeble respiratory murmur, or by a complete absence of all respiratory sound. When the effusion is moderate, there is egophony; when it is more considerable, the vocal resonance is simply feeble. The exceptions are not rare, especially in children, in which a distant bronchial respiration and a bronchoegophony are noticed.

The *treatment* of primary acute pleurisy is very analogous to that of primary acute pneumonia. In the early stage, if the

constitution is vigorous, the fever considerable, and the pulse active, you must have recourse to free venesection, carried to the point of faintness, which may be repeated several times even, in severe cases, until the febrile symptoms abate. To venesection you should add the free use of leeching or cupping to the side affected. In a great majority of the cases which enter this Hospital, the most active period of the disease has passed away. Copious effusion has taken place, and the febrile symptoms are moderate; while the stitch in the side is the chief subject of complaint. In such cases, it is not always advisable to practise venesection, or, if resorted to, it should be used with moderation. Free local depletion is, then, a much better remedy, producing more relief and less exhaustion than venesection. With other methods of depletion, you may properly combine the use of the tartar-emetic in the same way as I recommend you to use it in the treatment of pneumonia. The value of the tartar-emetic in the treatment of acute pleurisy, consists in its evacuant and sedative influence in overcoming febrile excitement and its consequences. As soon as this is accomplished, I prefer the use of mercury to any other remedy, and especially calomel, combined with antimonial, or Dover's powder. The value of mercurials in the treatment of serous inflammations is generally acknowledged, and this value consists not only in the power they possess of controlling inflammatory action, but in their efficacy in promoting the absorption of effused lymph. This object, although of less moment in pleurisy than in some other diseases —iritis or endocarditis for example—is yet of great importance, inasmuch as a lung must necessarily be crippled very much in its future action by thick and extensive false membranes.

After the constitutional effects of mercury have been induced, the absorption of the effused serum may be promoted by the use of diuretics: of these the iodide of potassium is one of the best. The carbonate of potassa with wine of colchicum, is also a useful diuretic. Digitalis has been much employed in these cases, and its sedative influence may sometimes recommend its use. It has been stated by a high authority, that even purulent effusion into the pleural sac might be absorbed, bodily so to speak, and carried off by the kidneys under the influence of digi-

talis. I have never witnessed such effects. On the contrary, I am inclined to believe that the influence of diuretic remedies on an inflammatory effusion, even if serous, is but moderate in degree. When the inflammatory action is fairly subdued by antiphlogistic treatment, the powers of nature seem generally disposed to produce an absorption of the effused fluid.

It is in this more advanced stage of the disease, as well as in the more chronic cases, that blisters applied to the side affected produce good results. They probably act by removing any lingering inflammatory action, and by stimulating the absorbents.

It is a point of great importance in the treatment of acute pleurisy, that the patient should be confined to the bed; and not only that, but that the chest should be kept carefully covered from the external air. There is reason to believe that in inflammations so near the surface as pleurisy, the external air coming in free contact with the chest may act unfavorably. French practitioners are in the habit of enveloping the affected side in a poultice, especially in the treatment of children; and this practice, if care be taken to keep the poultice warm, is often advantageous. A portion of oiled silk, fitted to the affected side, is also beneficial in excluding the external air. But none of these means will avail any thing unless the patient is kept in bed, and is carefully covered by the bed-clothes. It is the more necessary to mention this fact, because patients are not always very sick with pleurisy, and are often disposed to sit up, at least in bed, while the disease is still in an active state; or if in a more serious condition and tormented by the restlessness of fever, they are equally liable to exposure.

The diet and regimen proper in acute pleurisy is that which is appropriate to all inflammations; absolute diet, as perfect rest as possible. By absolute diet, I mean the use of diluent drinks, with occasionally a little arrow-root or gruel. But all cases, even of acute pleurisy, are not benefited by a rigid diet. Constitutional peculiarities, or complications, will often render a more nutritious diet proper; and it must never be forgotten that cases do occur where a mixed treatment—supporting the general strength while you subject the patient to the influence of local depletion, or of mercurial action, is plainly indicated.

The rapidity with which effusions from acute pleurisy are absorbed is not very great. When the effusion is moderate, you may expect a more rapid improvement. When dilatation of the affected side exists, with other evidences of copious effusion, the progress is usually very slow; weeks or months may elapse before you can admit that all effusion has disappeared. Even then, a degree of dulness on percussion, and a feebleness of the respiratory murmur remains, and very often will remain, perhaps for life.

Patients often complain, after a severe attack of pleurisy, of more or less pain or uneasiness, or of a sense of weariness in the lower portion of the affected side after fatigue or exposure. There is for a long time a weak spot, about which the physician may be consulted. Time and the use of stimulating frictions, especially with cold water, are the best remedies for this condition.

The practice of forcibly inflating the lungs by an inhaling-tube, or by other means, appears to me an appropriate treatment in cases of pleurisy, when all symptoms of inflammation have subsided. All practitioners are aware, that one great difficulty to contend with in these cases, is the want of expansion of the compressed lung. As long as the lung is compressed and inactive, so long a serious obstacle exists to the removal of the effusion. In cases in which the lung is firmly compressed by adhesions, so that the opposite surfaces of the pleura cannot come in contact and adhere to each other, the effused fluid will not be absorbed. It will gradually pass into a purulent condition, and empyema and its consequences will be the result. But if the lung can be made to expand when the effusion begins to be absorbed, this expansion will hasten the progress of absorption, and tend to restore the lung to its natural condition, and diminish the chances of a contraction of the chest.

The value of opium in the treatment of acute pleurisy is much greater than in the treatment of pneumonia. In the latter disease, you will be obliged to use it with care, for fear of checking expectoration. But in pleurisy, as, indeed, in all acute serous inflammations, opium, sometimes in large doses, is highly beneficial.

When you meet with a case of chronic pleurisy, in addition to other signs of copious effusion, you will commonly find the affected side dilated. The chances of success in producing an absorption of the effusion depends very much upon the fact whether this condition exists or not. In cases in which, notwithstanding the universal dulness on percussion, the form of the side affected remains natural, it is a good omen. It is a still better omen, when you can detect a certain degree of resonance on percussion beneath the clavicle.

The treatment now generally pursued in this Hospital in cases of chronic pleurisy, is that recommended by Dr. Hope, about ten years ago. The fundamental points in his mode of treatment are, a generous diet, and even tonics, combined with mercurials, more or less freely used, according to circumstances, and carried to the point of salivation. He also recommends blisters and diuretics, but regards them as of minor importance. If hectic symptoms exist, this does not contra-indicate, necessarily, the mercurial treatment. These symptoms may be combated by the mineral acids.

By some practitioners, the mercurial inunction to the skin over the side affected is regarded as the preferable mode of exhibiting mercury.

But if all remedies fail, and these cases are by no means rare, if the effusion obstinately continues, and the patient is daily sinking under the combined influences of oppression and hectic, the question of opening the pleura and of discharging the matter, treating the case like an abscess, naturally presents itself to your consideration.

There can be no doubt that many lives have been saved by the spontaneous, or by the artificial discharge of the purulent collection.

There is no doubt also, in my own mind, that many cases in which the discharge of pus occurred at a late period, and in which death finally ensued, that recovery would have taken place, had the discharge of the purulent secretion taken place at an earlier period in the disease.

My decided impression is, that in all cases, after proper remedies have been tried in vain, the operation for empye-

ma should be resorted to, and, if possible, before the vital powers are much exhausted. Because, notwithstanding the great and immediate relief experienced from the discharge of the pus, still, a great deal of it remains to tax the powers of life. A more or less copious purulent secretion continues often for a long time.

There are three classes of cases, in which the question as to the propriety of performing the operation may be discussed.

First, there are the cases in which the side is much dilated, the intercostal spaces bulging and fluctuating, and in which pointing, even, has occurred. These are the cases in which the operation has most generally been performed. Before the discovery of auscultation these were the only cases in which it could be performed with propriety, for in such cases only could the existence of matter in the pleural sac be ascertained with any degree of certainty. Many cases, in which the operation is performed under such circumstances, recover, but death is by no means of rare occurrence. The patient is relieved, often very much relieved at first, but he soon dies of exhaustion.

Again, there is a class of cases in which the disease, having resisted all treatment, presents a different condition of things. The affected side is not at all dilated, or but slightly so; the intercostal spaces may be a little dilated, or not, but there is no fluctuation, and especially no pointing. Shall an operation be advised in this case? I think so, and that the chances of success will be greater than in the first class of cases. There is one thing that you must endeavor to be certain about—that is, the actual existence of pus in the chest. The history of the case, the progress of the physical signs, must be your guide, and your judgment must guard you against a hasty decision.

Finally, there is another class of cases, in which the effused pus has been absorbed partially, and in which the dilatation of the affected side, if it had existed, has given place to even a partial contraction. Yet the existence of great dulness, and the absence of a respiratory murmur over the lower portion of the lung, the existence of hectic fever, and of other symptoms, must lead you to believe that the pus is still there, and that it refuses to be absorbed. The cause of this cessation of the absorption is proba-

bly the compressed state of the lung, which refuses to expand. What shall be done in this case? Open the chest? I confess that I have never seen the operation performed under these circumstances, but I have examined fatal cases in which I wished it had been performed.

Another question presents itself in these cases. What is the condition of the lungs? What is the condition of other organs? It is certainly desirable to know that the lungs were probably healthy before the attack, and that no evidence exists of any subsequent disease. Suppose, after examining the chest, you suspect that an abscess is forming in the lung? Shall this make you hesitate? Shall you wait and see if nature will not open a communication with a bronchus, and thus discharge the pus? You may wait in vain for this result, and even if it should, in time, occur, it is a far less agreeable and thorough mode of evacuating the chest than the operation of paracentesis. Suppose that you have reason to fear that the patient may be tuberculous, should that deter you? I think not. Would you not open an abscess anywhere else in a tuberculous patient? Would the discharge of pus exhaust him? I think not. It would relieve him, and thus prolong his existence.

Even in cases in which a softened tubercle has ruptured into the pleural cavity, and a bronchial communication has been, at the same time, established, constituting what is called hydro-pneumothorax, should this operation even then be performed? I have never seen the operation performed under these circumstances but once, and then a fatal termination soon ensued. But I have recently met with two cases in this Hospital, in both of which the post-mortem examination made me hesitate as to the propriety of the course pursued. In one case, all the signs of hydro-pneumothorax continued until death, yet after death the lungs were found so nearly healthy, the tuberculous deposit was so small, that I could not help thinking, had the operation been performed—this was decided against in consultation—the life of the patient might have been prolonged, and his condition rendered more comfortable.

Another case occurred, in which the signs of hydro-pneumothorax existed, but after a time the evidences of communication

with a bronchus ceased, and this condition continued until death. In this case, also, the lungs were very little diseased, and the opening into the bronchus could not be detected by inflating the lung. It had no doubt been closed, perhaps by being covered by a coating of lymph. In this case, and for a still stronger reason, the bronchial communication having ceased, the operation might have aided materially in prolonging life.

It is difficult to say what the precise condition of the lung is in such cases. But this we do know, that hydro-pneumothorax occurs most frequently, when there are but few tubercles in the lung. A copious deposit of tubercles leads to a secondary pleurisy with effusion of lymph only, by which the cavity of the pleura tends to become obliterated, and the form of disease I am now considering is no longer likely to occur.

The operation for empyema is very simple, and is easily performed. The space between the fifth and sixth ribs, where the serratus magnus muscle digitates with the external oblique muscle, is the point recommended by Laennec. If, however, fluctuation can be felt lower in the chest, it will be proper to make the opening there. I have opened the cavity of the pleura as low as the space between the tenth and eleventh ribs. When pointing occurs in the upper portion of the chest, between the second and third ribs, as frequently happens, fluctuation, or at least other evidences of pus, can be detected in the lower portion of the chest. It is better, I think, in such cases, to open the pleural sac as low as the space between the fifth and sixth ribs. Then the accumulated pus will more readily escape.

A difficulty that I once knew to occur in performing this operation was quite singular. A gentleman had all the symptoms and physical signs of empyema. The operation was decided to be proper. A trochar was introduced, but no pus followed the operation. A probe pushed forward through the canula to its whole length, met with no obstruction. The operation was abandoned. But, two or three weeks after this attempt, pointing occurred at the precise spot where the operation was performed, and a spontaneous evacuation of a large quantity of pus took place. The patient died, and the cause of the failure of the operation was quite apparent on post-mortem examination.

A dense and firm false membrane, like buckskin, lined the whole pleural cavity. It adhered but slightly to the subjacent pleura; so that when the trochar was pushed forward into the chest, it pushed this membrane before it without penetrating into the cavity which contained the purulent fluid.

The mode of performing this operation belongs to surgery. I may, however, state the manner in which I have generally performed it. The patient sitting up in bed, or in a chair, the skin is drawn down with the thumb of the left hand, and an incision, about two inches in length, is made along the upper edge of the sixth rib. This incision may be continued nearly through the intercostal muscles. A trochar may then be passed into the sac of the pleura, care being taken to keep as closely as possible to the upper edge of the sixth rib, and to obeerve that one of the flat surfaces of the instrument is directed upward. By adopting these precautions, you will avoid the danger of wounding the intercostal artery. Some surgeons prefer opening the pleura with a common abscess-lancet; and this method answers very well when pointing has taken place, or when superficial fluctuation is felt. In cases, however, in which the pressure of the pus has not yet produced this result, and the parietes of the chest retain their natural thickness, the trochar is, I think, the better instrument.

Experience proves, that the admission of air into the chest in these cases, is attended with no serious inconvenience. If care be used, a very considerable portion of the purulent fluid can be evacuated before any air enters. It is, perhaps, better, when air begins to pass through the canula, to withdraw the instrument, and allow the skin to fall over the opening. A poultice may then be applied, the patient placed in bed lying on the affected side, and an opiate administered. The relief which follows the operation is usually very great. The patient sleeps much better than before, his night-sweats abate, and his pulse diminishes in frequency. The subsequent treatment should consist in the proper administration of opiates, of tonics, and in the use of nutritious diet—care being also taken that the opening in the chest shall not close spontaneously, especially during the early progress of the treatment. The daily use

of a probe, or, if necessary, of a small tent, will prevent this accident.

A large proportion of the cases thus treated recover, if the operation is not delayed too long. About two-thirds of the cases that have fallen under my observation have terminated successfully, and this has been especially the case when the operation has been performed on children.

LECTURE XI.

LARYNGITIS.

Acute primitive laryngitis.—Œdema of the glottis.—Croup, or membranous laryngitis.—Spasmodic, or false croup.—Causes.—Symptoms.—Treatment.

When acute laryngitis terminates fatally, the morbid appearances you will observe on dissection are by no means uniform. The mucous membrane, which may be regarded as the original seat of the disease, is far from presenting, in all cases, the chief consequences of the morbid action. This membrane, indeed, will be found to have lost more or less of its natural polish, and to be somewhat thickened and softened in its tissue; often this thickening, as in other cases in which the mucous membrane is inflamed, occurs in patches varying in size. Sometimes, indeed, the surface presents a granular appearance. The softening of the mucous membrane, like the thickening, is apt to be partial, and in those portions of the larynx most exposed to attrition from the passage of mucus, this membrane may be actually rubbed off, so as to produce abrasion, and, finally, ulceration. You will most frequently notice these appearances upon the internal edges of the vocal chords. You will also notice more or less redness of the mucous membrane—sometimes diffused, more frequently occurring in patches, and accompanying the evidences of inflammatory action already mentioned. This redness, sometimes bright and vivid, sometimes indistinct, will be found, as in cases of bronchitis, to be chiefly seated in the submucous cellu-

lar tissue. You will often observe distinct red lines or points, formed by the partially filled capillary vessels; and at other times a diffused blush, seated, perhaps, more directly in the mucous membrane itself.

You will find the surface of the mucous membrane, in these cases, covered by a viscid, tenacious mucus—sometimes mucopurulent in its character, and, perhaps, tinged by blood.

At other times, especially in children, the surface of the mucous membrane is covered by an exudation of lymph, constituting the form of disease, properly called *Croup*. This exudation is sometimes abundant, lining the whole cavity of the larynx, and presenting a line or more of thickness. In other cases, it occurs in patches—sometimes very thin and delicate in appearance. The adhesion of this exudation to the mucous membrane varies also in different cases. This adhesion may be very intimate; in other cases it is very slight, or it has even become detached from the subjacent parts, being separated from them by a cream-like fluid, perhaps, purulent in its character.* This separation of the exudation may occur in large masses, so as to present a cast of the subjacent parts; but this is most apt to occur in the lower portion of the larynx, and especially in the trachea. You will notice the more delicate and more closely adherent patches in the upper portions of the larynx, and especially about the vocal chords, where they are less easily separated, and become detached in small portions.

It is common, in all the forms of acute laryngitis, for the mucous membrane, both above and below the larynx, to exhibit the same traces of inflammation. More or less decided inflammatory action will be formed in the throat, in the trachea, and even in the bronchi, in certain cases. These inflammatory changes of the mucous membrane, whether they consist in redness, thickening and softening, or in the secretion of mucus or of pus, or in the exudation of lymph, are in all important respects similar to those noticed in the larynx. Indeed, it seems very certain, that in a large proportion of cases, the first inflammatory changes take place in the throat, and extend downward

* Cheyne regarded this a lymph in a liquid form.

to the larynx; while in other cases, much more rare no doubt, the affection commences in the trachea, or even in the bronchi, and extends upward. Cases, however, do occur occasionally, in which the disease begins in, and is confined to, the larynx, and even to a limited portion of the organ, as, for instance, to the epiglottis.

The inflammatory changes, in cases of acute laryngitis, are by no means confined to the mucous membrane. The submucous cellular tissue is commonly affected, and often in a way to lead to the most serious consequences. You will remember the peculiar attachment of the mucous membrane to the subjacent cartilages by means of this tissue. About the root of the epiglottis, in the folds of mucous membrane which pass from the arytenoid cartilages to the epiglottis, and downward, below the ventricles, as far as the cricoid cartilage, this attachment is very loose, and the cellular tissue, the bond of union, of course is very abundant. While below the ventricles, over the cricoid cartilage and the rings of the trachea, the attachment of the mucous membrane to the subjacent cartilages becomes suddenly very close and intimate. This loose submucous cellular tissue becomes readily infiltrated with serum, or with a sero-purulent secretion, which rapidly distends the mucous membrane, so as more or less completely to fill up the cavity of the larynx, and of course to prevent the passage of the air. This distention is in part mechanical, for the close attachment of the mucous membrane below, prevents the effusion from diffusing itself. This accumulation of fluid in the submucous cellular tissue, commonly described as *œdema of the glottis*, is by no means necessarily connected with considerable inflammatory action. Indeed, the swelling is sometimes so pale as to have led to the doubt whether any inflammatory action, at all, has existed.

In certain cases, you will find lymph effused into the submucous cellular tissue, instead of serum, and in other cases, especially if the disease has been severe and somewhat protracted, the cellular tissue is infiltrated with pus. In the cases of purulent infiltration, the mucous membrane may lose its vitality in certain portions, and small eschars may form, by which the pus may escape into the cavity of the larynx, the subjacent cartilages

and muscular tissue being exposed, and more or less disorganized.

In severe cases, serum, or lymph, or pus, may be discovered in the cellular tissue external to the larynx, and the cervical glands in the neighborhood will also frequently be found inflamed.

The lungs in fatal cases of laryngitis do not readily collapse, and the bronchi are filled with frothy mucus. Pulmonary œdema, and not unfrequently evidences of pneumonia, or of pleurisy, will also be noticed.

The chief *cause* of acute laryngitis is exposure to cold and moisture, the ordinary cause of inflammation of the air-passages. Laryngitis, when it assumes the form of croup, is sometimes epidemic. Erysipelas of the head and face, typhus fever, as well as the eruptive fevers, especially measles and small-pox, predispose to laryngitis as a secondary affection; other cases are owing to the direct application of irritating causes. The inhalation of steam, an attempt to drink boiling water by mistake, have caused the disease; an accident apparently not unfrequent among the poor class in England. Acute laryngeal attacks are certainly more common in children than in adults, and apparently more frequent among males than females. There is a constitutional predisposition in some families to these affections, especially to that milder form of disease known as the spasmodic or false croup; and the same fact is probably true to a certain extent of the membranous or true croup.

The more severe form of acute laryngitis is a very rapid and fatal disease. It is ushered in by a rigor, by a sensation of lassitude, and by other febrile symptoms accompanied by a slight soreness of the throat, and by some difficulty in swallowing. These symptoms are soon followed by a difficulty in breathing, by a short, dry, hoarse cough, with pain, tenderness, and a sense of stricture in the region of the larynx. The voice also becomes hoarse or stridulous. The difficulty of respiration rapidly increases, and soon becomes alarming. The countenance is flushed and anxious, the ability to swallow much impaired or quite lost, and the hoarse cough followed by an access of strangulation. The respiration in these cases becomes stridulous, and the difficulty with which the air enters the lungs is most marked

during the act of inspiration, especially in those cases in which much œdema exists.

This œdematous condition of the larynx, so important to be recognized in reference to the treatment, is usually readily ascertained. By passing the forefinger into the throat, the epiglottis can easily be explored, as well as the upper portion of the larynx. In cases of œdema, this valve will be found swollen and erect, and the loose tissues constituting the arytenoid-epiglottic folds will be felt distended and swollen, filling up more or less completely the upper orifice of the larynx.

Cases may occur in which the œdema is confined to the ventricles of the larynx, and is beyond the reach of the finger, but such cases I think are rarely met with.

It will be noticed in cases in which this œdema of the larynx exists, that the difficulty of inspiration is much greater than that of expiration. The explanation of this is simple. The bags of serum formed by the arytenoid-epiglottic folds are sucked into the larynx during inspiration, and forced out again during expiration. In these severe and dangerous cases, unless the patient is speedily relieved, the tendency to strangulation increases. The countenance becomes pale and livid, the anxiety and restlessness increase, the pulse becomes feeble and much accelerated, the extremities grow cold, delirium and coma ensue, and the patient expires sometimes very suddenly.

Cases of œdema, still more dangerous, may occur in which no evidence of the affection can be discovered by the touch—cases in which the disease affects more particularly the parts about the ventricles of the larynx. Or in other cases, the submucous effusion, instead of being fluid and serous, may be solid and composed of lymph. The important practical point I wish now to insist upon is this: that in many cases, the obstruction is owing to an œdematous swelling of the upper portion of the larynx that can easily be detected by the touch, and be promptly remedied and life saved by a free scarification of the parts affected.

It may happen also, that the epiglottis is the chief seat of the disease, and even fatal cases of this kind have been recorded. In these cases the chief symptom is an utter inability to swallow. There is also frequently pain in protruding the tongue,

and a sensation as if a lump was in the throat, accompanied by a distressing hawking and the difficult expectoration of a viscid mucus.

In the milder form of laryngitis, occurring principally in children, and called the catarrhal, the false croup, there is seldom any imminent danger. There is, in particular, no tendency to œdema, nor to any form of effusion into the submucous cellular tissue. In fatal cases, the mucous membrane of the larynx presents the ordinary appearances of inflammation, viz., redness, swelling, softening, with increased mucous secretion. Evidently in these cases, a chief condition of the parts affected escapes entirely all anatomical observation. This is a tendency to spasm of the glottis. Indeed, in a large proportion of cases the evidences of inflammatory action are very slight. Sometimes, indeed, they are entirely absent.

This subacute form of laryngitis, this false croup, is sudden in its attack. It may be preceded by a slight soreness in the throat, hoarseness and cough, like ordinary bronchitis. There is seldom much febrile disturbance. The child goes to bed and falls quietly asleep in its usual way. But during the night, it is suddenly awakened in great distress by a violent paroxysm of dyspnœa. The stridulous or sonorous inspiration, the hoarse, barking cough, point at once to the larynx as the seat of obstruction. With these sudden and violent symptoms, although the face may be flushed by excitement, the skin remains comparatively cool, and the pulse, although excited, is neither full nor active. After a time the paroxysm will subside, and the child, perhaps, fall asleep. But soon, during the act of coughing, a new paroxysm supervenes, and all the symptoms return in their aggravated form, not, however, so severely as at first. This may be repeated several times, each paroxysm being milder than its predecessor, until at length the patient remains quiet, breathing, perhaps, with a little noise and oppression of the respiration, and awaking in the morning a little hoarse.

These paroxysms may be repeated the succeeding night, but in a less degree, if the case is neglected; but in most cases, the disease is entirely controlled by very simple remedies.

This blending of the effects of slight inflammatory excite

ment with spasmodic action of the glottis is not entirely confined to children. Adults are sometimes subject to the same sort of attack, and in certain families this tendency is particularly strong. It is in hysterical females that these cases are most apt to occur.

Although in most cases, a certain amount of inflammatory irritation in the fauces or in the larynx seems to precede these paroxysms of laryngeal dyspnœa, I do not think that this is always the case. In nervous persons an attack of indigestion has sometimes appeared to me to be the exciting cause. Agitation of mind may perhaps produce the same result.

In children more particularly, sudden attacks of stridulous inspiration, or crowing respiration, as it is sometimes called, occur, which seem entirely sympathetic—a purely nervous affection of the larynx. These cases have been most frequently referred to the irritation of dentition. Sometimes they have their origin in cerebral irritation, and are then apt to be associated with spasm of other parts of the body. They have also been supposed to be dependent upon the pressure of tumors in the neighborhood of the larynx, inducing irritation or paralysis of the recurrent laryngeal nerves. An enlarged thymus gland, pressing upon the front of the trachea, will induce the same symptoms, and may even lead to a suddenly fatal result.

The following case occurred to me several years ago: An infant boy, seven months old, fat and stout, had been subject from his birth to a slight cough, and to attacks of strangulation excited by the cough. These attacks had of late grown more frequent, occurring five or six times during the twenty-four hours, less frequently, however, at night. The child would grow blue in the face, struggle for breath, and recover itself again—each paroxysm continuing about a minute. During the intervals, the child was perfectly well, except the cough; there was no dyspnœa, no gastric or intestinal derangement. One evening the child was sitting upon its mother's knee, lively and well, when one of the paroxysms occurred, and in a minute the child was dead. The fauces, larynx, trachea, and bronchi were perfectly healthy; the lungs were less crepitant than usual, but healthy. The heart was somewhat enlarged; but the chief mor-

bid appearance was an hypertrophy of the thymus gland, which was four inches long, and three inches broad, reaching from the middle of the trachea to the diaphragm. The nerves of respiration, especially the par vagum, did not appear to be implicated.

Similar cases of sympathetic irritation of the larynx, inducing, as I suppose, spasm of the glottis, occur in the adult from the pressure of aneurismal tumors on the trachea.

A case of this description has recently occurred in this Hospital, which illustrates very well the leading phenomena of this interesting affection.

A sailor about thirty years of age, entered the New York Hospital in great distress for breath, and quite exhausted. I saw him a short time after his admission. He was then sitting upon the side of the bed, leaning forward, with his elbows upon his knees. The countenance was pale, the extremities cold, and the pulse extremely feeble. The dyspnœa was considerable, the respiration stridulous, especially in expiration. There was dysphagia, and a hoarse, barking cough. The patient referred all his distress to the larynx, and to a sense of obstruction there. His voice was not materially affected. An examination of the fauces, of the epiglottis, and of the upper opening of the larynx, discovered nothing. Externally there was no swelling, no tenderness over the larynx. The distress of the patient was much aggravated by the examination. The dyspnœa and the stridulous respiration increased, and the voice was entirely lost. After a few minutes' rest, the respiration became much more easy, and the voice returned—sometimes being feeble and indistinct, at other moments quite natural.

The patient stated, that for four months past, he had had some cough and dyspnœa, with shooting pains in the right side of the chest. His laryngeal difficulty had commenced only five days before his admission; at that time, he supposed his throat was sore. During their continuance, his distress had, at no time, been very great, except on attempting to take exercise. The morning of his admission, he had attempted to cross the street to go to a barber-shop, when so violent an attack of strangulation occurred, that he fell insensible in the street, and was obliged to be carried into the house.

The absence of pain or tenderness in the region of the larynx, of inflammation of the fauces; the non-existence of any mechanical obstruction, so far as the sight and touch could determine, the remarkable aggravation of the symptoms after exercise, and their improvement after a few moments' rest; the position of the patient, he preferring the sitting posture and inclining forward, as giving him the most comfort; the extreme feebleness of the pulse, quite out of proportion to other symptoms, led me to suspect the existence of an aneurism pressing upon the trachea. A careful examination, however, was of no avail, not a single physical sign of aneurism could be detected; but in examining the neck, a slight crackling sensation from external emphysema of the cellular tissue was observed. The respiratory murmur was feeble all over the chest, the sound on percussion was natural.

The condition of the patient did not materially alter until he died, two days after his admission. He had no more paroxysms of strangulation, his voice was for the most part natural, he swallowed without much difficulty, he coughed occasionally. He could sleep lying on the left side, but the pain in the right side prevented his assuming that position, and if placed upon his back, he would at once rise up and lean forward, saying that he was suffocated. He was found asleep during the night, sitting up in bed, with his head resting upon his knees. He died from gradual exhaustion.

The fauces and the larynx were quite natural. An aneurism of considerable size, communicating freely with the upper portion of the descending aorta, and containing very few clots, pressed upon the trachea just above its bifurcation. The vessel itself was disorganized by chronic inflammation. The lungs were œdematous, and contained a few tubercles in progress of cure. The heart was healthy.

Cases of laryngitis, attended by the formation of false membrane in the larynx, occur most frequently in children, and especially before the age of six years—resembling in this respect the false croup which I have just described. The disease is sometimes witnessed in the adult, in connection with erysipelas, or in the course of chronic diseases. This membranous inflam-

mation is to be distinguished from the other forms of acute laryngitis by an examination of the throat and by the detection of false membranes upon the mucous membrane of the fauces or tonsils. It is a fact incontestably established, that in membranous inflammation of the larynx, the throat is similarly affected, and, as in other forms of laryngitis, the throat is the primary seat of the inflammation. Rare and exceptional cases no doubt occur, in which the disease commences in the larynx, and never reaches the fauces. It is also probable, that the attack may commence in the trachea and extend upward to the larynx. In these exceptional cases, you will possess no certain means of distinguishing this membranous inflammation from an acute attack unattended by this exudation. In both cases, there is the same febrile excitement, the same characters of the cough, voice, and respiration—the hoarse, ringing cough, and tone of the voice, the hissing or sonorous inspiration and expiration, with a gradually increasing dyspnœa, the paroxysms of strangulation, especially after coughing. Yet there is one symptom which may remove your doubts, but it occurs only at an advanced period of the disease, and that is, the expectoration of false membrane. This may, indeed, not occur in cases in which the membrane exists, or it may be swallowed, or otherwise escape observation. It has been said, that the existence of partially detached false membrane may sometimes be recognized in the larynx by auscultation, by detecting a flapping sound during the passage of the air through the organ.

It is common, in the different forms of laryngitis, for a rattle, produced by mucus or other secretions, to occur in the larynx and trachea, especially as the disease progresses. It requires no stethoscope to detect this. The ear applied near the throat of the patient, will readily detect it. It is a favorable indication; and, in cases of membranous inflammation, it is the time to look for the expectoration of the false membrane. This membrane may be expectorated in small shreds, but is sometimes thrown out in larger quantities. I have known a perfect tube, four inches in length, making a complete cast of the trachea and of its bifurcation, to be expectorated. This expectoration is usually attended with much relief, as indeed you might expect. It

diminishes the mechanical obstruction to the passage of air. But the relief is often only temporary. New membrane is formed, or the lungs have become so much implicated, or the nervous energy is so much exhausted, that the child finally perishes.

There are, then, three varieties of acute laryngitis which deserve your particular attention. First, the simple form, attended by the secretion of mucus, and often terminating fatally by the supervention of submucous effusion of a serous or of a sero-purulent fluid, sometimes of lymph or pus. The tendency to submucous effusion, in these cases, does not appear to depend upon the severity of the inflammation. Indeed, in some cases, the evidences of inflammatory action are so slight as to have classed this affection as an œdema, and to have led some to suppose the effusion to be dropsical, or the effect of venous obstruction, rather than of inflammation. The pale, bladder-like swellings you will sometimes notice after death, might deceive you, were there not evidences during life of inflammatory action, at least in the neighborhood of the swellings. My own impression is, that the inflammation which most frequently induces this submucous effusion is of the erysipelatous form, while in other cases, it occurs when the constitution is under the influence of depressing causes, as, for instance, during the convalescence of typhus fever.

The second variety of acute laryngitis is croup, and is attended with the exudation of false membrane. Most commonly it is a disease of childhood. It is sometimes epidemic, and is, in a very large proportion of cases, a fatal disease; probably not more than one-fifth or one-sixth of the cases terminate favorably.*

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* The mortality of croup, as compared with the general mortality, gives:

New York	(period of	3	years),	1 in	53 deaths.
Boston,	"	5	"	1 in	43 "
Baltimore,	"	4	"	1 in	31 "
Charleston,	"	5	"	1 in	122 "
London,	"	1	"	1 in	103 "

In the city of New York, during 3 years:

620 cases occurred from December to June.
347 cases occurred from June to December.

Of these, 523 were males, 444 females.

In the city of London, during 1 year:

249 cases occurred from December to June.
189 cases occurred from June to December.

It is sometimes met with in adults. Erysipelas, when it attacks the larynx, is sometimes attended with the exudation of lymph, and I have seen several cases of croup in the adult, occurring in the course of chronic disease; as phthisis, for example. The leading fact to be remembered in this form of inflammation is, that the exudation of lymph commonly occurs in the fauces, before it attacks the larynx. This is a fact of great diagnostic value.

The third variety of laryngitis is the very common and easily cured affection known as the spasmodic or false croup. This variety constitutes, by far, the largest proportion of the cases improperly called, in common language, croup. It occurs, like true croup, most frequently in childhood, and in certain families. It is, however, sometimes met with in the adult. My own impression is, that in most cases, there is slight inflammation of the mucous membrane of the larynx, but that the chief difficulty is produced by a spasm of the glottis, dependent upon a peculiar nervous irritability of the parts. I do not, however, suppose that any degree of inflammation, however slight, is necessary to induce this spasm. Remote causes, acting on this natural irritability, may induce it; as, for instance, indigestion.

The treatment of acute laryngitis may be successful by the use of the ordinary antiphlogistic remedies, unless permanent mechanical obstruction to the passage of the air occurs, either from effusion into the submucous cellular tissue, or from the exudation of lymph upon the free surface of the mucous membrane. If either of these conditions exists, the best-directed antiphlogistic treatment will fail. The first duty of the practitioner in these cases is to examine the throat, and the superior opening of the larynx, and discover, if he can, whether any such obstruction exists. If they do not, antiphlogistic remedies, active according to the circumstances of the case, should be at once resorted to. If the patient be vigorous, and much febrile excitement be present, he should be bled freely and even repeatedly from a vein, and leeches should be applied near the seat of the disease, although not directly upon the larynx. The tartarized antimony should also be administered in nauseating doses, and mercurials, especially in the form of calomel. In delicate constitutions, in

those who are convalescing from prostrating diseases, or are suffering from actual disease that has exhausted the vital powers, these active remedies are not to be employed except with great moderation and caution. In every case, this antiphlogistic treatment, unless speedily followed by relief, should lead you to a new examination of the upper portions of the larynx, to discover if possible whether an œdematous swelling of the parts has occurred. If this has occurred, antiphlogistic treatment can no longer be of avail, the obstruction has become mechanical, and must be remedied by mechanical means. The best practice, heretofore, has been to open the larynx or trachea below the seat of the obstruction. But fortunately, a much more simple, and in many cases, a very successful mode of treatment has been introduced. Three or four years since, Dr. Buck introduced into this Hospital the practice of scarifying the mucous membrane of the larynx, in cases of œdema—of scarifying, especially, the distended folds which pass from the arytenoid cartilages to the epiglottis. This scarification, if it penetrates to the submucous cellular tissue, will discharge the serous or sero-purulent effusion, not only from the part to which it is applied, but from the surrounding parts. The distention, and of course the obstruction to the passage of air, is at once diminished, and the relief experienced, in favorable cases, is rapid and decided. Still the œdema may return, and require new scarifications. The case, therefore, must be carefully watched, and proper antiphlogistic and soothing remedies must not be neglected. In many cases, certainly, the inflammatory action that produces the œdema is very slight, and probably disappeared when the effusion had taken place, so that a single scarification may be quite successful.

This mode of treatment is not entirely new; it was practised by Lisfranc, and occasionally by other surgeons; but the practice has not been generally recommended by practical writers on the diseases of the larynx. It is even ridiculed by Ryland in his treatise on this subject.

The ease with which the upper portions of the larynx can be explored by the finger, and the facility with which a scarificator, properly constructed, like that invented by Dr. Buck, can be carried along the finger used as a director, can only be appre-

ciated by those who have resorted to this mode of exploration and practice. It is as simple, as it is, often, effectual.

In certain cases, but these, I think, are rare, the œdema is confined to the parts about the vocal chords. It is, of course, then, impossible to be certain of its existence, or to reach it, if it were known to exist, by scarification. In other cases, the effusion into the submucous tissue, instead of being serous and fluid, is solid, being formed of lymph. In these cases, whether the obstruction can be detected by the finger or not, scarification can afford no relief. It may be doubtful, from the examination with the finger, whether the swelling is produced by a fluid or by a solid substance. But, provided a swelling can be distinctly felt, scarification should be at once resorted to, and the degree of relief obtained, often speedy and decided, becomes diagnostic of the character of the effusion, and indicates, moreover, the necessity, or otherwise, of a more decided step—the performance of tracheotomy.

In membranous laryngitis, or croup, the antiphlogistic treatment—venesection, leeching, antimonials, and especially mercury, is that usually resorted to. But it may well be questioned whether this mode of practice is really attended with much benefit. Certainly, most cases go on, without even a temporary improvement, to a fatal termination; and in the few cases that recover, it is doubtful whether the treatment has contributed much to the favorable result. It has been thought that the free exhibition of mercurials is beneficial, by favoring a change in the character of the inflammation, by promoting a secretion of muco-pus. In this way it is thought that the effusion of lymph may not only be controlled, but that the secretion of muco-pus favors the separation and expulsion of the membranes already formed. It is far from being true that the disposition to form false membranes is dependent upon the violence of the inflammation. Many cases of croup occur in feeble subjects in whom there is but little constitutional reaction, and post-mortem examination confirms the opinion that the local inflammation has been very moderate in degree. There can be no doubt, also, that many cases, which have yielded to the prompt and judicious use of antiphlogistic remedies, were cases of simple acute

laryngitis without the formation of membrane, a form of disease attended by symptoms quite similar, in most respects, to those of croup; and in children, not apt to be attended by submucous infiltration, and quite curable in its nature. But when false membranes exist in the throat, and you have therefore reason to believe that they have extended to the larynx, the influence of curative means is far less decided. For myself, when I look into the throat, and find nothing there except, perhaps, the traces of simple inflammation—in children, certainly—I am inclined to hope for a favorable issue; but if, on the contrary, false membrane is noticed, the prognosis at once becomes very unfavorable, and antiphlogistic remedies will avail but little. So unavailing has been the efforts of some of our best practitioners to cure genuine croup by antiphlogistic remedies—or by a perturbating treatment, consisting in the frequent administration of emetics in conjunction with antiphlogistic remedies, that some have thought best to abandon this mode of treatment and to leave the case pretty much to the efforts of nature, administering anodynes, especially small and repeated doses of Dover's powder, and permitting the inhalation of the vapor of warm water, rendered emollient by medication. This mode of treatment, while it has not, thus far, diminished materially the mortality of the disease, is said to have been favorable to the prolongation of life, and to have been especially conducive to the comfort of patients, by diminishing the frequency and violence of the paroxysms of strangulation, which are so formidable and distressing.

But are there no mechanical means to relieve the obstruction, analogous to the scarificator in cases of membranous effusion? There is reason to hope, and some reason to believe, that the application of the nitrate of silver to the larynx, 40 to 60 grs. to an ounce of water, by means of the whalebone and sponge, may be attended with benefit, especially in cases in which the inflammatory action has originally been inconsiderable, or has been subdued by other means. There is reason to think, that this remedy not only acts favorably upon the mucous membrane, but that this mode of application may be made useful by aiding, mechanically, in the removal of loose or of partially detached

membranes in the larynx. It is a mode of treatment fairly entitled to a trial before resorting to tracheotomy. Still, you must recollect, what post-mortem examination teaches you, that the false membranes in the larynx are not only more delicate, but more closely attached than to other portions of the mucous membrane. You must remember, also, that these false membranes usually extend into the trachea, and sometimes even into the bronchi, beyond the reach of mechanical means of relief.

Thus, it must be evident, from the greater difficulty of applying mechanical agents to the larynx in croup, owing to the age of the patients usually affected, and the inability, in many cases at least, to apply these means to the seat of the obstruction, that the use of the whalebone and sponge does not promise the same advantages as scarification in cases of submucous effusion, in which the superior age of the patient and the size of the parts enable you to manipulate with more ease and certainty; and when, if the whole extent of the obstruction cannot be reached, yet, from the free communications of the cellular tissue, the swelling of the parts beyond the reach of the scarificator may be effectually diminished.

But suppose that these different means of removing laryngeal obstruction fail in affording relief. Suppose that in simple laryngitis the submucous infiltration is chiefly about the vocal chords, beyond the reach of scarification, or that the effusion is solid in its character; suppose that the lymph upon the mucous membrane is too closely attached to be removed—then nothing remains to be attempted but tracheotomy. In cases of submucous effusion, the chances of relief from tracheotomy are very great, because the obstruction does not extend much below the vocal chords—the trachea is free. But in membranous inflammation, the chances of relief are far less, because the false membranes often extend into the trachea, and are, indeed, often very thick and abundant in this situation. The operation of tracheotomy, therefore, frequently fails in affording much benefit to the patient.

Remembering that the operation of tracheotomy is simply called for to remove an obstruction in the larynx, which has become permanent and is beyond the reach of antiphlogistic remedies, and which, moreover, cannot be reached by simple

and less formidable means—when these facts are well established in your minds, you should not delay the operation a single hour, if it can be avoided. Many times, this operation, although the case was perfectly suited to it, has entirely failed, because it has been deferred until too late. The imperfect oxygenation of the blood, in these cases, acts unfavorably upon the brain, producing congestion; and upon the whole nervous system, producing exhaustion. It acts also unfavorably upon the lungs, causing serous effusion into the cells. Thus, although the operation may afford, for the moment, entire relief, yet the patient dies.

There is a distinction, I think, to be made in these cases. When the progress of the disease has been comparatively slow, and the symptoms of exhaustion have gradually supervened, the chances of benefit are indifferent, if this condition is fairly established—that is, if the patient is drowsy, the pulse very much accelerated and feeble, and the skin cold. But when the patient is, at first sight, in a much worse condition, even apparently dead from a sudden attack of strangulation early in the disease, and before the system has become involved, even then, the operation of tracheotomy, if rapidly performed, and artificial respiration be at once established, the patient may speedily revive, and in half an hour be not only very comfortable, but free from danger.

In the cases which are of such frequent occurrence, especially at night, of spasmodic contraction of the larynx, the prompt administration of an emetic is often attended by complete relief, especially when indigestion is the cause of the attack. In the cases, also, in which there is slight inflammatory irritation of the larynx, the relief experienced after an emetic is also very great. But it is not complete. Such cases require further treatment, especially by the use of small doses of the tartar-emetic, of ipecac, or of the compound syrup of squills. Warm pediluvia, low diet, confinement to the house or to the chamber, must also be enjoined, until the laryngeal symptoms have disappeared. If the process of dentition is going on, the gums should be examined, and lanced if swollen, and the digestive organs should be carefully attended to. If, finally, the condition of the cere-

bral functions and of the nervous system, generally, lead you to believe that the laryngeal spasm is dependent upon the condition of the brain, the case must be regarded as a cerebral disease, and the treatment appropriate to that disease must be adopted.

LECTURE XII.

CHRONIC LARYNGITIS.

Anatomical characteristics.— Causes.— Symptoms.—Varieties.—Treatment.— Polypi of the larynx.—Foreign bodies in the larynx and trachea.—Pulmonary congestion; pulmonary apoplexy and œdema.

Chronic laryngitis is, in a great majority of cases, a condition of the scrofulous diathesis. In most instances, indeed, it is associated with manifest and advanced tuberculous disease of the lungs. In other cases, the evidences of pulmonary disease are not so well marked, or there is no evidence of their existence until, in the progress of the case, they begin to appear. Other cases which recognize a local exciting cause, as undue exercise of the larynx, or in which the disease has been propagated from the fauces to the larynx, appear to me to be generally associated with the scrofulous constitution. If you inquire carefully into the family history of such patients, and into their antecedent diseases, you will find, I think, abundant evidence to support this conclusion. It is not in Hospital practice that such inquiries can be made with any advantage. It is in the circle of private and of consultation practice that you can arrive at the conclusion I have stated.

This important fact should be deeply impressed upon you. When you see a case of chronic laryngitis, the idea that tubercles exist in the lungs should at once suggest itself to your minds. Even if evidences of their existence are not clear, their presence may be suspected, or, at least, feared.

But are there no cases of chronic laryngitis unconnected with the existence or the suspicion of tuberculous disease? There are

certain cases, in which the disease is evidently syphilitic. You may see fifty cases of tuberculous or scrofulous laryngitis, without meeting with a single case that is clearly venereal in its origin. Yet such do occur, and in which the disease exists in its most marked form. There are, besides, affections of the larynx, as the formation of polypi, certain diseases of the cartilages which, in their progress may induce chronic laryngitis, although originally independent of it. Finally, there are cases, and these are not infrequent, in which some of the symptoms of laryngitis may be present, especially loss of voice, but in which there is no real inflammatory action existing. In these cases, a loss of nervous power in the organ seems to be the chief morbid condition.

The organic changes in chronic laryngitis are often very simple. They consist chiefly in a thickening of the mucous and of the submucous tissues, sometimes united with induration of these tissues. Sometimes the mucous surface is more red than is natural, from partial injection, but more frequently the color of the tissue is a dead grayish-white. It is the effect of inflammation to destroy the natural polish of the mucous membrane; hence you will find its surface tarnished and dull, with a loss of its natural smoothness. Sometimes, indeed, it is granulated, or elevated in irregular patches. Finally, a more or less abundant mucous secretion will be noticed, sometimes adhesive, and closely attached to the mucous membrane; at other times muco-purulent in its character. I do not think that these simple cases pass readily into ulceration, unless neglected, or unless the lungs become affected by tuberculous disease.

Ulceration of the mucous membrane of the larynx is of frequent occurrence in connection with tuberculous disease of the lungs, and is chiefly found in connection with advanced cases of that disease; that is, with the existence of cavities in the lungs. These ulcers may exist upon any portion of the mucous membrane, but they are most frequently found at the posterior junction of the vocal chords, upon the vocal chords, and upon the laryngeal surface of the epiglottis. They are usually isolated, but are sometimes more numerous, and even confluent. They tend to penetrate into the subjacent tissues, sometimes

having for their base the thickened, grayish, or tawny submucous tissue; at other times, exposing the softened and disorganized muscular tissue. The mucous membrane in the immediate neighborhood of these ulcers is usually very little altered—a little tarnished, perhaps, nothing more; so that these ulcers do not seem to depend upon a general inflammation of the mucous membrane, but probably upon a very limited morbid action. Sometimes, however, the ulceration is more diffused, sweeping off the mucous membrane, and exposing the subjacent parts in an inflamed and disorganized condition.

Syphilitic ulceration is more destructive than tuberculous ulceration. It spreads over more surface, it tends to disorganize the subjacent parts more frequently. Reaching the cartilages, it produces ulceration of these bodies, changes rarely met with in tuberculous cases, although occasionally noticed in the epiglottis.

The disorganizing tendency of chronic laryngitis attended with ulceration, is exhibited in the ligaments of the larynx, which are ulcerated or softened, and blended with the cellular tissue. Ulceration may also extend to the articulations of the cartilages, producing dislocation. The laryngeal muscles are sometimes found softened, wasted, disorganized.

Chronic laryngitis is sometimes a secondary affection, dependent upon antecedent disease, or at least upon disease external to the mucous and submucous tissues. Thus, abscesses may form in the cellular tissue external to the cartilages. A favorite seat for them is in the cellular tissue, between the cricoid cartilage and the pharynx. It has received the name of *post-cricoid abscess*. This collection of pus disorganizes the cricoid cartilage: it produces necrosis or ulceration. It opens into the cavity of the larynx, and inflames and disorganizes the mucous and submucous tissues. Thus, a most severe form of chronic laryngitis is induced.

There is an affection of the cartilages of the larynx, the cause of which is not very well understood, occurring chiefly during the early period of adult life, which also produces a most severe and fatal form of chronic laryngitis. Imperfect bone-like matter is deposited in the cartilage, which is incapable of organization, and which, increasing gradually in extent, at last acts as

a foreign body, causing ulceration and suppuration of the surrounding parts. It is the cricoid cartilage, which is the chief seat of this disorganization. When the disease has terminated fatally, this cartilage will be found more or less degenerated into a calcareous substance lying loose in the centre of an abscess, which has communicated with the interior of the larynx, and perhaps also with the pharynx, while the interior of the organ presents a thickening or ulceration of the mucous membrane, with other changes common in chronic laryngitis.

Finally, organic changes occur in the cavity of the larynx, which, although not originally inflammatory, yet lead to chronic laryngitis as a secondary result. Thus polypi form in the cavity, forming tumors, sometimes pendulous, and which obstruct and irritate the organ. Or the epithelium becomes the seat of disease, and vegetations or even tumors form, which equally become a source of secondary laryngitis.

I have expressed the opinion, that most cases of chronic laryngitis occur in connection with the scrofulous constitution. But there are exciting causes. Thus, in many instances, you can trace the disease to the undue exercise of the voice during repeated attacks of catarrh, connected with sore throat, and with slight hoarseness. An affection, which, under the influence of rest, would probably subside in a few days, is thus continually aggravated, until at length it becomes a permanent malady. It is very common among clergymen. An impaired state of the general health, dyspeptic symptoms, mental anxiety, and fatigue, no doubt aggravate very much the morbid condition of the parts, and, probably, in many cases, are instrumental in its production. But the most frequent cause of this laryngeal inflammation is, undoubtedly, actual disease in the lungs and the secretion of purulent matter, which, by coming in contact with the laryngeal surface, and in subjects constitutionally predisposed to inflammation in these parts, induces ulceration of the mucous membrane; or in some cases, perhaps, only a thickening of the tissues. There can be no doubt, also, that the mere act of coughing, or the undue exercise of the voice, often plays a conspicuous part in the production and in the aggravation of the laryngeal symptoms in tuberculous subjects. Thus, when the cough is

more violent, the laryngeal symptoms, especially the hoarseness, are much aggravated. Sometimes, indeed, they disappear entirely for a time, when the cough abates, although the pulmonary disease remains as before. In this respect, tuberculous cases resemble the more simple form of the disease, at least when ulceration is not present, which I am far from thinking a necessary consequence, even in tuberculous subjects.

In most cases, when chronic laryngitis exists, the fauces are also affected, and it is common to refer to them as the primary seat of the disease. It is common to hear of the inflammation spreading from the fauces to the larynx, and so downward to the bronchi, and finally to the lungs, inducing disease of the lungs. The real truth is, I think, that in the majority of instances, the lungs were first affected, and that the disease has spread upward to the larynx and to the fauces. The simple irritation of coughing, either with or without expectoration, will induce laryngitis, in a mild form very likely, and produce also a similar affection of the fauces. You can hardly look into the throat of a tuberculous patient without finding evidences of chronic inflammation,—more vascularity than is natural, and the mucous membrane irregularly thickened. This is, then, a secondary affection, the consequence of pulmonary disease. In other cases, indeed, the throat may be the primary seat of the disease, and it may travel downward to the larynx, and, perhaps, to the bronchi, but I know of no evidence that it will produce disease of the lungs. This affection of the throat will sometimes follow an attack of scarlatina, and the patient, long afterwards, be subject to acute inflammatory attacks from trifling causes, and which terminate, at length, in a permanent or chronic inflammation of the parts, and which may eventually extend to the larynx.

The influence of dyspepsia, with an impaired state of the general health, in continuing, if not in causing the slighter forms of laryngeal disease, is very striking. In these cases, the throat is usually inflamed, and sometimes ulcerated, and the larynx seems to participate in the same difficulty. Yet many of these cases are by no means attended by important organic changes in the larynx. I am inclined to think that weakness and relaxation of the parts

more directly concerned in the production of the voice, is often the chief difficulty, and will explain the hoarseness and the sense of exhaustion that frequently follow the exercise of the voice in clergymen; and that the good effects of stimulating applications to the parts, combined with rest, is quite as much owing to their increasing the nervous energy, as in removing any traces of inflammation that may be supposed to exist. This loss of nervous energy is also, I think, sometimes a cause of the temporary hoarseness you will so frequently meet with in the earlier stages of tuberculous disease of the lungs, and which is apt to occur after much coughing, and especially in the evening, after much talking during the day.

It is difficult to say, in any case, whether, or not, a certain amount of inflammation exists in cases of slight laryngeal disease, when it exists without decided disease of the lungs. It is probable that there is some congestion, and, perhaps, some thickening, about the vocal chords. But when the disease has occurred after repeated and undue exercise of the voice, accompanied by a feeble state of the general health, dyspepsia, and other indications of mental and of physical exhaustion, I am decidedly of the opinion, that a loss of nervous energy in the parts affected is the chief thing to be considered.

Symptoms of chronic laryngeal disease are sometimes induced by quite an opposite condition of the system and of the parts affected. A state of general plethora, and of local congestion probably, about the neck, will induce many of the symptoms of chronic laryngitis. I remember the case of a clergyman, a robust, plethoric man, with a short neck, in excellent general health, and withal a free liver, who was affected with loss of voice, and with the symptoms of laryngeal obstruction, after an unusual effort in speaking. He tried various remedies without success, but was at length cured permanently by the repeated application of leeches to the neck. Similar cases in plethoric, free livers may occur without any particular exercise of the voice, and occasion much distress and anxiety. I have thought that the mechanism of these cases was similar to that existing in the case of an aneurism, and that external pressure from turgid blood-vessels upon the trachea, was the true cause of the symptoms.

The symptoms of chronic laryngitis resemble, in many respects, those noticed in the acute form of the disease. When the epiglottis is chiefly affected, difficulty in swallowing is the leading symptom. In cases in which this portion of the larynx is much crippled in its action from thickening, rigidity, or, still more, by ulceration which has destroyed a considerable part of its substance, the dysphagia is sometimes distressing, and the patient is nourished with difficulty, owing to the regurgitation of his food. It is remarkable, however, that in cases in which the epiglottis has been nearly destroyed by ulceration, if the ulcers are healed, the power of swallowing is sometimes so much improved that but little inconvenience is experienced, a proof that the mere destruction of the epiglottis has not so much influence on this function, as the pain and swelling produced by actual disease.

When the vocal chords are the chief seat of the disease, the voice is principally affected, becoming feeble, hoarse, stridulous, cracked, or sinking into a whisper. When ulceration has destroyed a considerable portion of the vocal chords, you will notice the most decided effects on the voice—it is permanently, irretrievably lost. In milder cases, a slight degree of hoarseness only exists, and this may even be hardly noticed, except when the voice is much exercised, or after violent coughing. In these cases, there is probably nothing more than a slight thickening of the mucous membrane about the vocal chords, and perhaps hardly that; for, in many cases where hoarseness is a slight and variable symptom, it is more probable that it depends upon a want of nervous power in the organ, rather than upon any organic change.

When there is obstruction to the passage of air through the larynx, dyspnœa exists, and the respiration becomes noisy, stridulous. The patient feels that every act of respiration costs him an effort, and that the air, as it passes through the larynx, is obstructed in its course. The inspiration, particularly, becomes sonorous and prolonged, and this, perhaps, is more distinctly marked than in the expiration, although, in very many cases, there is no perceptible difference. Exercise, of course, aggravates this difficulty, or any cause that excites the circulation.

This affection of the respiration, this laryngeal respiration, as

it may be called, is usually permanent, in different degrees, and when it exists, indicates an organic obstruction in the larynx. But there are exceptions to this rule. Serious disease of the lungs, as emphysema, may induce a respiration, in which dyspnœa and sonorous inspiration and expiration are prominent symptoms. So may a tumor pressing upon the trachea. Yet in these cases, the symptoms want some of the characteristics of laryngeal disease, and a careful inquiry will often enable you to form a clear diagnosis. When the larynx is the seat of obstruction, the patient refers his distress in breathing to that organ. He places his finger upon the upper portion of the thyroid cartilage. There is his difficulty, his stricture. The presence of pain, of tenderness on pressure over the larynx, will often aid you, and occasionally you will derive assistance from stethoscopic signs.

Patients suffering from permanent obstruction of the larynx are liable to sudden and violent paroxysms of suffocation from spasm. This distressing accident does not necessarily happen, even in the worst cases of chronic laryngitis, neither is the permanent obstruction always most marked in such cases. Extensive ulceration of the larynx, especially of the vocal chords, may, by destroying these chords, lessen the chances of spasm, and enlarge rather than diminish the passage for the air. It is thought, also, that effusion of serum into the submucous cellular tissue may suddenly occur in chronic laryngitis, and thus induce great dyspnœa. This is probably true, but I do not think that this is a common accident. It is more rare than you might expect it to be.

Chronic laryngitis is almost always attended by cough. Sometimes the cough has nothing peculiar in its character. It may be dry and tickling, especially early in the disease. It may be loose, and attended with expectoration. But in many cases, especially when the disease has affected the voice and the vocal chords, the cough is hoarse, often barking, spasmodic—it is a laryngeal cough. The expectoration varies very much in the different stages and in the different varieties of chronic laryngitis. When the disease is slight and in its early stage, there may be only a trifling expectoration of mucus, or hardly that.

But when ulceration exists, and the tissues of the organ have become disorganized, the expectoration may become abundant, purulent or muco-purulent, and stained with blood. A case of hemorrhage from the larynx has never occurred to me. Unless the attending symptoms were very decided, I should always suspect that it came from the bronchi, and that it was connected with tuberculous disease of the lungs.

Pain, tenderness on pressure, a sense of dryness, of heat, and constriction in the throat, are common symptoms in chronic laryngitis. There is, also, often a sense of weariness and of weakness referred to the organ. I do not know how far these symptoms are indicative of any particular condition of the larynx. But when pain and tenderness are permanent and limited, if a purulent or muco-purulent expectoration existed, I should suspect ulceration. The sense of weariness belongs rather to an atonic, or impaired nervous condition of the parts.

Auscultation is of great value in the diagnosis of chronic laryngitis; not the direct application of the stethoscope to the larynx—that is seldom of much use. The larynx is so superficial that the sounds generated in it are usually heard distinctly at a moderate distance from the organ. It is thus, that you will detect the sonorous or sibilant respiration, or the harsh sound of the air as it passes and repasses the larynx. Some observers have thought that, by the application of the stethoscope, they could detect the precise seat of the laryngeal disease, by observing a limited roughness of the respiratory sound, or a limited mucous rattle. But the stethoscope, or rather the direct application of the ear, is much more valuable in these cases when applied to the chest, as you may thus establish the existence, or otherwise, of disease of the lungs, a point of prime importance in all cases of chronic laryngitis.

External deformity occurs in cases in which the disease is primarily in the cartilages of the larynx, or external to this organ. An abscess pressing upon the organ may push it out of place, or produce swelling in its neighborhood. When an abscess forms between the cricoid cartilage and the pharynx, an unusual feeling of elasticity is said to be communicated to the touch, if the cartilage is pushed backward; also if the carti-

lage is rubbed against the spine, the natural feeling of solidity is wanting.

The most simple form of chronic laryngitis is when the disease succeeds an ordinary catarrh, being induced, for the most part, by a careless exposure to the vicissitudes of the weather. The patient complains of some soreness of the throat, which is often inflamed to a moderate degree, of tenderness over the larynx, of a sensation of heat and constriction in the part, while there is some dysphagia, hoarseness, slight sibilant respiration, with a laryngeal cough, perhaps attended by mucous expectoration. The patient may be of a vigorous and robust constitution, and although somewhat reduced, perhaps, if the disease has continued for a considerable period of time, yet with no decided or well-marked constitutional symptoms. Such cases, in which a slight degree of inflammatory thickening of the mucous and submucous tissues is the chief organic change, will, even after several months' continuance, yield readily to a mild course of mercury. It is in such cases particularly, that a local treatment, as the application of a solution of the nitrate of silver, forty to sixty grains to the ounce of water, and applied to the interior of the larynx by means of the whalebone and sponge, as first recommended by Trousseau and Belloc, may be permanently useful.

A second class of cases, far more numerous, and in part at least dependent on a constitutional cause, *the scrofulous diathesis*, are much more insidious in their development, and much more difficult of cure. The subjects of this form of this disease are pale and delicate persons, who inherit a feeble constitution. If a careful inquiry be made, it will frequently be noticed that such persons have, for a long time, before any distinct laryngeal disease has manifested itself, been subject to an irritation of the throat, indicated by a more or less frequent desire to clear it of mucus, and a tendency to slight hoarseness and weakness in the chest, after singing, or other exercise of the voice. In clergymen particularly, in whom this form of disease is very frequent, you will see the best type of its development. An examination of the throat discovers the parts somewhat congested, the tonsils slightly enlarged, the posterior fauces presenting

red, elevated patches of thickened mucous membrane, with increased mucous secretion. Yet, with these evidences of chronic inflammation, the parts are relaxed, the vessels are large and often arborescent, the uvula swollen, œdematous, and flabby, and capable of being drawn out to twice its natural length by moderate traction with the forceps. The degree of hoarseness presents every possible variety, while the sibilant respiration is rarely met with. The pain in swallowing, as well as the tenderness over the larynx, is seldom marked. The sensation in the larynx is like the aching from fatigue. Such cases are often much relieved by rest, by local applications, like the nitrate of silver; but they are very apt to return again, especially under the influence of unfavorable exciting causes, as undue exercise of the voice, fatigue, anxiety, and other depressing influences. This form of the disease cannot properly be separated from the constitution that is almost uniformly found to attend it, and the chances of permanent benefit are intimately connected with constitutional remedies which improve and invigorate the general health, such as a sea-voyage, travelling, freedom from fatiguing and anxious occupation, and the use of a general tonic course. Such patients are often encouraged by even a slight improvement in their condition. They even think that they are almost or quite cured, until a relapse teaches them that a perfect recovery is not so easily attained.

In these cases, you may discover no pulmonary disease. There may even be no cough, but only a hawking from the throat of mucus, although cough also is frequently present. Yet if, in time, the lungs do become implicated, which is very probable, and cavities form in them, then the laryngeal symptoms become aggravated by the supervention of ulceration. This is not, however, always the case, for the laryngeal disease, even if ulceration be present, may be masked to a certain extent by the pulmonary complication. Besides, much will depend upon the seat of the ulcerations. Those which attack the epiglottis give the most trouble, from the dysphagia they induce, while, if the vocal chords are attacked, the hoarseness may be much and permanently increased.

There are peculiar symptoms resulting from the *elongation of*

the uvula. This appendage sometimes touches the root of the tongue and the upper portion of the larynx, so as to create a frequent cough of a dry, irritating character. But at other times, the elongated uvula, especially at night, and when the patient is asleep upon the back, gets into the cavity of the larynx and induces spasm. The patient awakes suddenly in great distress, with a sonorous respiration, and with a sense of suffocation, which is relieved by the erect position, but sometimes only by passing the finger into the throat and drawing the enlarged uvula out of the larynx. Astringent and stimulating applications to the part will often reduce its size, for it is generally only relaxed and flabby in its structure, or a portion may be removed by the scissors.

Syphilitic laryngitis, ulcerative in its character, may be distinguished by the ordinary symptoms of constitutional syphilis having preceded, or actually attending, the laryngeal disease. Syphilitic eruptions, nodes, nocturnal pains, ulcers in the throat, or the evidences of the ravages of ulcers, are commonly noticed. Such cases are found in those laboring under the syphilitic cachexia; but occasionally, cases are met with in which there have been no previous constitutional symptoms, in which nothing but the original, primary syphilis, and the laryngeal affection, are known to have existed. Such cases are obscure, because there is nothing in the laryngeal symptoms themselves to distinguish a syphilitic case from any other case. These cases require a syphilitic treatment: sometimes mercury, sometimes the hydriodate of potass, or sarsaparilla, mercurial fumigations with cinnabar, and other means appropriate to the treatment of a syphilitic patient. Even when the laryngeal disease is severe, the patient frequently recovers, at least as far as the destruction of the parts by ulceration will permit.

When extensive destruction of the larynx occurs from *original disease of the cartilages*, or from the extension of the destructive process to them from ulceration of the mucous membrane, or from external abscess, the constitutional symptoms are usually decided. There is emaciation and hectic, besides all the evils of an aggravated local disease; the expectoration is ordinarily abundant in these cases, and purulent in its character.

Sometimes particles of gritty, bone-like matter are expectorated from the diseased cartilages; at times, also, the expectoration becomes highly offensive, being formed of pus that has been in contact with a carious, or mortified cartilage. Such cases are extremely distressing. Yet, while most of the prominent symptoms of laryngitis exist in an aggravated form, those arising from obstruction to the larynx may be less marked than in the more simple forms of the disease. The extensive disorganization of the tissues may leave the passage for the air quite as free as natural, unless when temporarily obstructed by the secretion of the parts, and for the same reason, the tendency to spasmodic contraction is diminished. Such cases are entirely beyond the reach of remedies. Palliatives may relieve the distress of the patient, as the inhalation of conium, the use of morphine, and sometimes tracheotomy may be called for.

There is an affection of the larynx, in adults, in which there is no inflammatory or other organic change present, and which is characterized by a single prominent symptom, *aphonia*. It seems to be dependent upon a simple loss of nervous power, and its most evident cause is long-continued exertion of the voice. I remember a street-crier, who, after the exertion of the voice for a whole day, at last could only speak in a feeble whisper; the nervous energy of the larynx was exhausted. In such cases, rest is the first indication of treatment, and if this is not successful, the inhalation of chlorine gas, and of other stimulating substances may be tried, also electricity, and the internal use of strichnine. These cases of nervous aphonia are sometimes associated with an irritable condition of the larynx, especially in nervous females. I remember a very striking case of this kind. A young lady of a highly nervous temperament had suffered from aphonia for several months. With it was associated a dry cough, consisting of a single expiration, but which was incessant, at least every half minute during the day. At night the patient fell asleep readily and remained undisturbed until morning, when the cough returned. This case was finally cured, after the trial of many remedies, by the internal use of strichnine. When aphonia is associated with hysteria, it may be attended by paroxysms of dyspnœa and by sibilant respira-

tion. There is seldom any complaint of pain, unless the attention of the patient is called to it; afterwards, it is often complained of.

Neuralgia of the larynx is sometimes met with as a severe and obstinate affection.

These nervous symptoms, referred to the larynx, require the aid of a constitutional and tonic treatment, and especially of mental diversion, when associated with evident hysteria. Indeed, all cases of purely nervous aphonia are likely to be benefited by constitutional remedies.

In every form of chronic laryngitis, rest is a most important point to be attended to. I mean rest of the affected organ. Many cases, no doubt, become chronic, or are aggravated beyond the hope of cure, by a disregard of this simple indication. The clergyman, the public singer, goes on exercising his voice, when his vocal organs should be kept as quiet as possible. The man of business rises in the morning comparatively well, but when night comes, after a day spent in talking, he is more hoarse, his cough is increased, and he passes a restless night. In this way, the disease is gradually aggravated; and even while means of relief are attempted, it is no unusual thing for a patient to continue the same pernicious habit of exercising the organ much more frequently than is desirable. The simple passage of the air through the larynx is, no doubt, unfavorable to a cure, for you will notice, in cases in which tracheotomy has been performed, and the patient breathes for a considerable time through a tube, that the effect of perfect rest to the organ is sometimes surprisingly beneficial. Indeed, after tracheotomy has been performed, local remedies can be applied with an advantage and a success that is quite remarkable.

Thus the *operation of tracheotomy* is not only useful when the patient is in immediate danger of suffocation, but it answers another important indication, by enabling the diseased larynx to be kept nearly in a state of rest. A current of air is no longer constantly passing and repassing over the diseased surface; the organ is no longer agitated by coughing, or by the attempt to speak. Still, tracheotomy has never been performed, except as a last resource, to relieve suffocation. It is not unlikely that it

may be employed under less threatening circumstances, to assist in the cure of the more obstinate forms of the disease.

There are two forms of laryngeal disease, both of which are of much interest, with which I wish to occupy your attention for a few moments at this time.

Polypi sometimes form in the larynx. They are seated upon different portions of the interior of the organ, perhaps most frequently upon the inferior vocal ligaments. They sometimes originate in the fibrous tissue, sometimes in the submucous cellular tissue, sometimes in the mucous membrane. Their structure depends a good deal upon their origin, being sometimes fibrous, when they are firm and resistant; or cellular, when they are less dense and more vascular; or mucous, when they are quite soft and pulpy. Frequently these three elements are united in the same tumor in different degrees; but whatever may be the leading element of its composition, it is always covered by mucous membrane. These tumors are usually pediculated, and movable to a certain extent in the cavity of the larynx. They commonly excite no inflammation in the organ, unless when they induce ulceration by pressure.

There is another form of tumor met with in the larynx, which is more superficial, having its seat in the epithelium of the mucous membrane, but penetrating, in time, more or less deeply into the subjacent tissues. These *epithelial tumors* are usually numerous, and often clustered. They appear like vegetations, and often resemble in appearance the venereal vegetations of the penis and of the parts about the anus. But they are not necessarily of this nature. Clustering in masses, they often form a tumor of considerable size, which may so completely fill the cavity of the larynx as to render tracheotomy necessary. These tumors may be also pediculated, so as to move in the cavity of the larynx.

The symptoms of polypi of the larynx are obscure. They act like foreign bodies in the larynx, and the symptoms they induce must depend much upon their precise seat, their size, and the degree of motion that their attachment will permit. In the best recorded cases, the disease commenced very insidiously, without pain, or other indications of laryngitis—except a gradually

increasing hoarseness followed by dyspnœa, and finally, by paroxysms of suffocation. Sometimes a sensation of something moving upward and downward in the larynx, like a valve, has been noticed, and, in one instance, where the tumor was seated upon the upper portion of the larynx, it could be felt by the finger passed into the throat. In a case which I saw some time since, with my friend, Dr. Buck, these symptoms were all well marked, and on opening the larynx the mucous membrane about the ventricles and the vocal chords were covered on each side by clusters of small polypous excrescences, some of them quite movable by their attachment to the mucous membrane, but forming, by their union, a considerable obstruction in the cavity of the larynx. There was also a tumor occupying the ventricles of the larynx, deeper seated, and composed of cellular and fatty matter.

It has been proposed, in these cases, in which a polypous growth is suspected, to divide the larynx through its whole extent, and even to extend the incision above, as high as the os hyoides, and below, to the upper rings of the trachea. By this method the cavity of the larynx can be thoroughly explored and a polypus removed. This operation has already been successfully performed, and is well worthy of being attempted in doubtful cases, both as a means of exploration and as a means of cure, if a polypus is found to exist.

Cancerous tumors are very rarely developed in the cavity of the larynx. They are not pediculated, they destroy the cartilages of the larynx and penetrate into the surrounding tissues, forming a tumor external to the organ. Their diagnosis is uncertain—they are beyond the reach of treatment. Tumors of a tuberculous character are still more rare, if indeed they have ever been found in the cavity of the larynx.

The *introduction of foreign bodies into the larynx* usually happens in this way: a child is playing with something in its mouth, a pin, a cherry-stone, or other substance, and is suddenly frightened or amused, and a strong inspiration at the unguarded moment carries the foreign body into the larynx. Or an individual, while eating, suddenly becomes excited to talk or to laugh, and a bit of cheese or a portion of a nutshell slips into the

larynx. The commotion that such an accident excites is usually most violent. The individual is instantly seized with a paroxysm of suffocation, and perhaps is dead before medical assistance can be obtained. Or the foreign substance may get into one of the ventricles of the larynx and thus create less permanent obstruction, while paroxysms of suffocation are constantly occurring with every fit of coughing, threatening life every moment, if not quite destroying it. The immediate performance of laryngotomy and the removal of the offending substance is clearly indicated in such a case. This may be pushed upward into the throat by a probe, or drawn downward through the external wound. The ventricles of the larynx should be especially examined. There is a case on record, in which the operation was performed in good season, but no foreign body could be found. The patient died, and the hook of a lady's dress was found in the ventricle, lying, in fact, just at the edge of the incision into the larynx.

In many cases the foreign body, after creating, perhaps, great commotion in the larynx, escapes into the trachea, and then the patient is much relieved, for a time, at least; or it may have passed at once through the larynx into the trachea. But if the body is light, so as to be carried up and down the trachea by the air, so as to strike against the glottis occasionally, it will induce paroxysms of violent coughing and of strangulation. Sometimes the foreign body can actually be heard striking against the sides of the tube, as it passes upward and downward. When the trachea is opened, in these cases, the foreign body is often expelled at once by the convulsive expirations that follow. Sometimes, if a pin be the offending substance, it will be found lying across the trachea, and it may be removed by a forceps.

Finally, the foreign body, if more heavy, finds its way down into a bronchus, and usually, from the anatomical arrangement of the first division of these tubes, into the right bronchus. The right bronchus, you will recollect, is a more direct continuation of the trachea than the left bronchus. So that a foreign body falling by its gravity, would naturally fall into the right tube. There are, however, exceptions to this rule, and I have met with a recorded case, in which the substance changed sides during its continuance in the air-tubes.

The immediate symptoms of a foreign body in a bronchus are not very urgent. There is cough, more or less dyspnœa, and the respiration over the lung affected is feeble, especially superiorly. The remote consequences are much more serious. Inflammation and abscess ensue, with purulent expectoration, hectic, and other severe symptoms, and the patient dies, sometimes after a lapse of one or two years, gradually exhausted. The patient, therefore, must be relieved of the foreign body, if possible. The feeble respiration, usually on the right side of the chest, and perhaps confined to the upper lobe, is the best guide to its seat. When the trachea is opened, it may be expelled by the current of air, or a long and narrow pair of forceps, or a probe bent like a hook at the end, may reach it, and enable the surgeon to extract it.

Other methods have been proposed to get rid of these bodies in the trachea and in the bronchi. It has been proposed to administer emetics, with the hope that the offending body might be carried upward through the larynx. I have pointed out the danger caused by a foreign body in the larynx, and there is great risk that, in attempting, a second time, to pass through this narrow and irritable passage, it will again be caught and induce fatal suffocation. Another method which has been tried with success, is to suspend the patient with the head downward, and allow the foreign body to pass through the larynx by the force of its gravity. This, of course, could only happen in cases when the foreign body is heavy, as a piece of coin. But here, also, there is danger of its being caught in the larynx. The best mode of proceeding, after all, seems to be to resort to tracheotomy.

Before I pass from the consideration of the inflammatory affections of the chest, I wish to call your attention to a condition of the pulmonary organs, which, although not inflammation, is sometimes closely allied to it, and may readily pass into it, in certain cases. I refer to *pulmonary congestion.*

Congestion is of two kinds, active or arterial, with a tendency to terminate in hemorrhage; passive or venous, with a tendency to terminate in effusion of serum. This distinction is a broad

and practical distinction. There are, no doubt, however, exceptions to the general rule. Passive hemorrhage may terminate, for instance, in hemorrhage, as in pulmonary apoplexy; and active congestion leads to the effusion of serum in the first stage of pneumonia.

Active or arterial congestion, probably, has its principal seat in the capillary vessels more immediately connected with the arteries. It is usually attended by constitutional febrile symptoms, not, however, in a very marked degree. There is increased heat, some acceleration of the pulse, and perhaps some dyspnœa, uneasiness, or pain in the chest, although these local symptoms are often entirely absent; unless relief is obtained, hemorrhage may ensue, and often a very serious hemorrhage takes place. This condition readily passes into inflammation: acute bronchitis, pneumonia supervene, and sometimes pleurisy.

Passive or venous congestion has its seat in the veins, and in the venous capillaries. It may be preceded by symptoms of oppression in the chest, but this is frequently absent. The pulse may be full and rather languid, but this is not necessarily observed. It terminates in the effusion of serum into the cavity of the pleura, constituting hydrothorax; or into the air-cells or the cellular tissue of the lungs, constituting pulmonary œdema: or it may terminate in pulmonary apoplexy.

Both these forms of pulmonary congestion are secondary affections: they do not attack, primarily, the healthy lung, unless, perhaps, in certain cases of asphyxia induced by hanging, or by the inhalation of carbonic acid gas. The chief cause is, the existence of tubercles in the lungs, or organic disease of the heart, especially that form of disease which commences in the mitral orifice of the heart, and which induces hypertrophy of the right ventricle. I cannot, therefore, speak to you of pulmonary congestion as an independent form of disease. I shall return to it again, when I speak of tuberculous phthisis and of organic disease of the heart. Still, I may properly describe now its pathological appearances and the prominent symptoms by which it is characterized.

The most simple form of pulmonary congestion is that noticed in the lungs of almost every patient who dies of any disease, either

acute or chronic, especially if the agony is somewhat prolonged. You will, almost always, in your post-mortem examinations, find the lower and posterior portions of both lungs in a condition resembling the first stage of pneumonia, only of rather a darker hue. These portions of the lungs contain more blood than is natural; they are more or less dark and unaerated, and with a considerable effusion of serum into the air-cells, and into the cellular tissue. This might, therefore, be regarded as a passive rather than as an active congestion, did not pneumonia in its first stage produce, apparently, the same condition. This condition, indeed, may be looked upon as a union of the two forms of congestion.

Pulmonary congestion is also the result of asphyxia; and in this case the first influence is felt by the venous capillaries of the lungs. The want of the due oxygenation of the blood seems to paralyze the venous capillaries, which should feel the stimulus of oxygenated blood; and congestion ensues, which extends to the larger trunks on the arterial side of the pulmonary circulation. You must remember, that in the lungs the natural order of the circulation is reversed. It is the artery which carries the unoxygenated or venous blood, while the veins transport the oxygenated or arterial blood. I once examined the lungs of a criminal who had been hung about an hour before the chest was opened. The lungs were large and crepitant, but they were uniformly of a darker color than natural. When divided by the scalpel, the pulmonary vessels were found filled with dark, blackish blood. There was no effusion of blood, or of serum. Had death been more gradual, serous effusion would, probably, have occurred. Indeed, the appearance of the lungs, when death takes place from gradual strangulation, is not the same. In addition to the congested state of the vessels, you will find effusion of serum into the air-cells and cellular tissue, and sometimes into the pleural sac; and, not unfrequently, with these evidences of congestion, you will notice traces of pneumonia, or of pleurisy. These are the appearances noticed in cases of laryngitis, and especially in croup.

The most frequent form of active pulmonary congestion is that which occurs in tuberculous disease of the lungs, and often in the early stage of the disease, when the pulmonary tissues

are compressed, and, at the same time, irritated by the deposit of miliary tubercles. This active or arterial congestion terminates in hemorrhage.

Another form of the hemorrhagic congestion is what is called *pulmonary apoplexy*. It consists in an effusion of blood into the air-cells, in the first instance, probably, and into the bronchi and the surrounding pulmonary tissues. It appears in the lungs in the form of hard, circumscribed, dark-red, or even blackish masses, granulated in their texture, and surrounded by pulmonary tissue, somewhat congested, perhaps, or quite healthy in appearance. These dark, indurated masses vary very much in size. Sometimes they are as small as a cherry-stone, sometimes they are as large as an almond. They are seldom larger than a goose-egg. In one instance, both lower lobes of the lungs had passed into this condition, but then, I think, it was blended with a certain degree of pneumonia, as it certainly was with pleurisy. As I have already remarked, these masses are granulated, and if scraped with the scalpel, after being divided, the granulations are easily changed into dark blood, leaving behind them little pits, or depressions. They are, evidently, little clots of blood filling the air-cells, and which are easily removed by scraping.

Sometimes these apoplectic masses are found to contain a cavity filled by a larger clot, and are produced by an evident rupture of the pulmonary tissue. Indeed, I think that Cruveilhier has recorded a case in which even the pleura was ruptured.

I have never examined these cases except when they have terminated fatally in a short time, and then the lungs presented the appearances I have now described. It is not, however, necessarily fatal. It would appear that the effusion is gradually absorbed, becoming more pale and yellowish, and more dry as this process progresses. The dark masses may excite sufficient inflammation around them to become inclosed in a cyst, or they may undergo a further change, and suppuration may ensue, and, perhaps, finally a cicatrix be formed. But I am speaking now of what I have never seen myself in the lungs.

Indeed, pulmonary apoplexy is, in my experience, a rare form of disease. During a residence of a year and a half in Paris,

where I was constantly in attendance upon the Hospitals, especially those in which chest diseases were most frequently to be found, I did not meet with a single case of the disease. Since that time, I have met with a few cases, and, I believe, always in connection with one uniform condition of the heart—viz., disease of the mitral valve, with hypertrophy of the right ventricle. These cases have always been attended by hæmoptysis, usually abundant, and followed by speedy death.

Laennec thought that these cases might be distinguished during life by physical signs. This great observer states, that you may detect an absence of the respiratory murmur over a small portion of the lung, and around this a crepitant rattle, which may not be of long duration; the former being caused by the small indurated masses, the latter by the surrounding serous effusion into the air-cells. Laennec says nothing about any dulness on percussion, limited in extent, as pointing out the seat of the apoplectic effusion. In Laennec's time, the same accurate results could not be derived from percussion as are now obtained by mediate percussion. Piorry, the inventor of this mode of percussion, and a most skilful practitioner of the art, states that he has discovered these indurated masses by the use of the pleximeter, but his opinion was not subjected to the subsequent test of a post-mortem examination. For my own part, I have never discovered any physical signs of pulmonary apoplexy; and in cases in which the indurated masses are of the ordinary size and number, and are surrounded by healthy lung, I do not believe that any physical signs of their existence could be detected, any more than when, in lobular pneumonia, a few scattered lobules of the lung are condensed by hepatization. I have already pointed out to you the difficulty of doing this in pneumonia. In pulmonary apoplexy, the general condition of the patient is unfavorable for a careful and prolonged physical examination. The attack of hæmoptysis, often severe and repeated, demands a state or rest which forbids auscultation, especially over the posterior portions of the chest. If, as I believe, these cases are always associated with heart disease, the physical diagnosis can be best ascertained by examining this organ, and this can be done without disturbing the patient in the least. If the physi-

cal signs of enlargement of the heart exist, and especially of the right side of the heart; if a blowing sound with the impulse of the heart is heard, principally over the apex, indicating mitral disease, the true character of the case may at once be suspected, and its previous history, especially the long existence of dyspnœa, and, perhaps, of palpitation—symptoms of heart disease—will enlighten you as to the true nature of the case.

Passive congestion of the lungs is most commonly attended by serous effusion. Sometimes this takes place into the pleural sac, constituting *hydrothorax*. This is also, as I have stated, a complication of heart disease, and, perhaps, more frequently of mitral disease. The cause of the congestion is obstruction to the return of the blood from the lungs to the heart. You may inquire how does it happen, that in some cases of the same disease, pulmonary apoplexy sometimes occurs, while, in other cases, a serous effusion is the result? I think, that in cases in which the right side of the heart is much hypertrophied, and in which the blood is sent into the lungs with unusual force in the attempt to overcome the obstruction on the other side of the heart, that pulmonary apoplexy will be the usual result; but that, in cases in which this hypertrophy of the right side of the heart is not so evident, and in which the action of the heart generally is feeble, that serous effusion is more common. But this is certainly true: they may both exist in the same case.

The detection of hydrothorax is by no means difficult. When in the course of a heart disease the respiration becomes more oppressed, you may find the cause of this increased oppression in the physical signs of serous effusion into the pleural sac, precisely, in fact, as in ordinary pleurisy. There is the same dulness on percussion, occupying especially the inferior and lateral portions of the chest on one side, and perhaps extending over the greater portion of the lung, with feebleness, or absence of the respiratory murmur, and perhaps egophony. But there is no pain in the chest, no febrile excitement, and, unless a previous bronchitis had existed, no cough. It seldom happens, I think, that hydrothorax is double at the commencement; but it usually becomes so before the close of life. Almost invariably, when I have opened the bodies of those who have died with this com-

plication, a more or less abundant effusion of serum has been found in both pleural sacs.

Hydrothorax is not a very frequent form of dropsical effusion in heart disease; neither have I very frequently found it in connection with Bright's disease of the kidney—a disease often attended by serous effusions, although the cause of this accident is entirely unknown. It is certainly less frequent than anasarca, or than ascites; but it is more frequent than effusion into the pericardium.

Pulmonary œdema is of two kinds: it may consist in an effusion of serum into the air-cells, or into the cellular tissue of the lungs. In either case, the lungs appear pale and glassy, unless they are injected by blood, as, indeed, often happens: they pit on pressure. When the air-cells are alone affected, if you cut into the lungs, you will observe, after moderate pressure, a gush of aerated serum. If, on the contrary, the effusion is into the cellular tissue, the serous effusion will not be aerated. Both these conditions are frequently associated.

This œdema, the result commonly of passive congestion, and of heart disease in its advanced stage, or of the advanced stage of Bright's disease of the kidney, when it appears to be entirely independent of congestion, is commonly slowly and insidiously developed, and often escapes observation, until it is discovered by an examination after death. In cases in which you might reasonably expect its existence, if you find at the base of both lungs —for the affection is commonly, I think, double—a slight dulness on percussion, and a feebleness of the respiratory murmur, without egophony, you may suspect its existence. These signs may, indeed, be owing to a limited hydrothorax; but generally in this case, the dulness on percussion is much more marked and extended. If the effusion is, on the contrary, into the air-cells, you may detect a fine crepitant rattle at the base of the lungs. There is the same danger of confounding this condition of the lungs with the first stage of pneumonia, as there is of confounding hydrothorax with pleurisy. The existence of heart disease, and the absence of febrile symptoms; or a characteristic rusty expectoration, and perhaps a cough, will establish the diagnosis, especially, if after a short time, no bronchial respiration exists.

In hydrothorax, however, if the lung is consolidated by previous disease, or if it is attached to the diaphragm, or to the ribs by old adhesions, a bronchial respiration may be developed, which might, without the consideration of other circumstances, lead to the suspicion that pneumonia existed.

Serous effusion into the air-cells is sometimes a very acute disease, and may terminate life in a short time. I have known it accumulate so rapidly and so abundantly as to flow out of the mouth even before death, and in only a few minutes after any urgent symptoms had occurred. In this case there was a moderate enlargement of the heart, and the air-cells and bronchi were full of serum. There was also considerable venous congestion. I shall not enter at this time into a consideration of the treatment of pulmonary congestion. Being a secondary affection, its treatment belongs to the disease in which it occurs. I will only remark, that active congestion requires an antiphlogistic treatment; blood-letting often, antimonials in nauseating doses, opium, the acetate of lead, perfect rest, and a cool atmosphere. While passive congestion, although sometimes benefited by moderate depletion, is most frequently benefited by rest and by local derivatives. When it terminates in dropsical effusion, purgatives and diuretics are indicated; and if hemorrhage occurs, small doses of the spirits of turpentine are sometimes very beneficial, as are also astringent remedies.

LECTURE XIII.

EMPHYSEMA OF THE LUNGS.

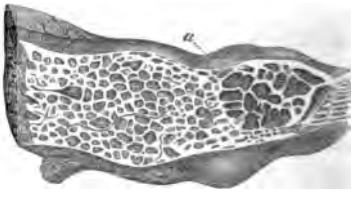
Anatomical characteristics. — Causes. — Symptoms. — Diagnosis. — Spasmodic asthma. — Cardiac asthma. — Diagnosis from tubercles in the lungs. — Treatment.

A LARGE proportion of the cases commonly described as asthma, an indefinite expression, embracing a variety of cases

quite different in their nature, are now recognized as connected with the anatomical change in the structure of the lungs, pulmonary emphysema.

This disease consists essentially in a dilatation, and commonly in an hypertrophy of the air-vesicles. It is probable, that in certain cases the walls of the vesicles may not become thickened at the same time that they are dilated. Analogy would seem to render this probable, and post-mortem appearances in certain rare cases may confirm this opinion. The general rule, however, is, that in emphysema the cavities of the air-vesicles are not only enlarged, but that their walls are thickened.

Indeed, when you open the chest of a person who has died of well-marked, general emphysema of the lungs, these organs will actually press forward out of the chest, as if its cavities were too small to contain them. The surface of the organ presents a grayish, shining aspect, and when carefully examined, it is covered with distinct vesicles, varying in size from a grain of sand to a pin's head, or to a pea. Usually, these vesicles are pretty equally developed, so as not to alter the general smoothness of the surface, but occasionally, large vesicles project from the surface. In most cases, especially if you examine the inferior edge of the anterior lobes, you will find this portion thick and blunt, or elongated by a kind of appendage resembling, sometimes in shape, the ear of a dog. This appendage is pale, even glassy, in its appearance, and when cut into is found to be composed of large, irregular cells (*a*), formed, evidently, by the union of many pulmonary cells in one cavity. The same appearance may also be found in other portions of the lung, but not so well marked. Sometimes, indeed, you will find in the substance of the lung, irregular cavities filled with air, which appear to be produced by a sudden rupture of the lung; and this opinion is confirmed by the fact, that they may contain traces of effused blood. Finally, in certain cases you will find air effused into the cellular tissue of the lung, especially into that portion directly under the pleura,



where it appears in flattened patches of considerable size, and movable under the pressure of the finger.

An emphysematous lung feels firmer than the healthy lung when pressed by the fingers, and when all the air is pressed out carefully, the pulmonary tissues feel more dense and thick than is natural. It crepitates less, but it floats more completely than healthy lung on the surface of water. In those portions in which the air-vesicles have united to form large, irregular cavities, the lung is very light and almost without crepitation, and shrinks immediately when divided with the scalpel.

There are no marks of inflammation in an empnysematous lung, except in the bronchi. These tubes present the usual evidences of chronic inflammation: you might expect to find them dilated, but this is not the fact. It is true, you may find evidences of pulmonary congestion, or of pneumonia, or of pleuritic adhesions, or of tubercles in the lungs in those who die of emphysema. Or an air-cell, or bronchus, may, by rupture, communicate with the cellular tissue, causing what is called interlobular emphysema. But these are accidental complications. One thing you will find pretty constantly, especially in advanced cases, and that is, enlargement of the heart. This, indeed, may be called the natural consequence and termination of emphysema.

After examining an emphysematous lung, you will see a ready explanation of all the appearances in the dilatation and hypertrophy of the air-cells. These cells finally, by pressure upon each other, produce an absorption of the intervening cell-walls. Thus many cells may be converted into a single cavity, producing a honeycomb appearance, when several such cavities are formed in the same portion of the lung. What remains of the old cell-walls may still retain its hypertrophied appearance, but this portion of the lung having become nearly or quite useless, and without function, these parts become rarified and atrophied, just as in the case of atrophy of a portion of the lung from obliteration of a bronchus: the walls of the cells become thinned, and finally absorbed, so that large and irregular cavities are formed in the rarified tissue at the expense of the air-cells.*

* In pulmonary emphysema, the first change is a simple dilatation of the air-

This genuine atrophy of the lung is, in its nature and progress, the very reverse of emphysema, although the latter disease, in a very advanced stage of its progress, and in certain portions of the lung, may bear some resemblance to it, or finally coexist with it.

Emphysema of the lungs may be partial or general. It is, probably, always partial at the commencement, affecting chiefly the anterior portion of the superior lobe. The lower and anterior edge of the superior lobe is, probably, the part in which the disease commenced. You will frequently find it there when it exists nowhere else, and in advanced cases it is usually most marked in this situation. It is here also that you will find, most frequently, those irregular cavities formed by the breaking down of the air-cells.

When the disease is partial, it is not unfrequently confined to one lung; but in advanced cases it extends to both lungs, and to every portion of them. It prefers, however, the superficial portions to those more deeply seated.

The influence of these organic changes exerts a great influence upon the circulation of the blood through the lungs. The first effect of the disease is to produce pressure upon the capillaries which ramify upon the air-cells, and, consequently, obstruction. But when the air-cells are broken up, the capillary vessels upon their walls must be destroyed with them. An emphysematous portion of lung, in its advanced stage, is anemic. Thus the circulation of the blood through the lung is gradually interrupted, and the effect of this is an enlarged heart.

It is difficult to understand the causes of emphysema in most cases. The disease appears to be hereditary. It is also con-

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vesicles. When many neighboring vesicles are dilated, they compress the intervesicular cellular tissue, and the vessels which it contains, until they disappear; at the same time that the air-vesicles, pressing more and more upon each other, finally break down into a common cavity, the size of which is proportioned to the number of vesicles which have united to form it. It may equal the size of a walnut, and presents, internally, partitions, or projecting spurs, formed by the remains of the cell-walls. The vascularity is also diminished in pulmonary emphysema. The air-vesicles are not hypertrophied. Their walls are rather thinned than thickened.—*Lebert.*

genital, or at least developed in very early life. Laennec thought that the explanation depended upon mechanical causes, and chiefly upon a dry catarrh, attended by swelling of the mucous membrane of the bronchi, with a viscid secretion of mucus. Thus the air admitted into the air-cells could not readily escape from them, would accumulate in the cells, and lead to their distension and hypertrophy. But this explanation of Laennec cannot be received. In many cases, the disease is developed entirely without any evidences of pre-existing bronchitis. It may exist in a well-developed form even, in certain rare cases, without any bronchial complication whatever. It is true, indeed, that bronchitis is an almost constant attendant of emphysema, but it is evidently the consequence, not the cause of that disease. There is something, certainly, in emphysema which disposes the bronchi to inflammation, and especially to spasm; facts which play a very important part, as you will soon perceive, in explaining the symptoms of the disease.

It is thought also, that the habit of playing upon wind instruments, and, perhaps, the habit of running, long continued, may predispose to the development of this disease. But the truth is, the most careful inquiry will not enable you to detect, in most instances, any appreciable cause.

The disease is, fortunately, very chronic in its progress, and can hardly be considered as shortening the average duration of life; although, in its advanced stage, it renders the sufferer from it an invalid, and, for the most part, incapable of business. In congenital cases, it often continues, with imperceptible increase, and with very little inconvenience to the individual, until perhaps the middle period of life. It aggravates the inflammatory affections of the chest, and renders them more fatal. It is not a preventive to the development of tubercles in the lungs. It leads directly, although slowly, to enlargement of the heart.

The most important symptom in emphysema is dyspnœa—*chronic dyspnœa*, with a tendency, as the disease progresses, to *severe and sudden paroxysms*. In congenital cases, the only symptom during the long period of childhood and of early adult life, may be a moderate degree of dyspnœa. Children affected

with this disease are easily put out of breath after running, and those who are older, suffer only after some unusual exercise, although there may be, constantly, a slight oppression, which is hardly noticed. But in time, paroxysms of dyspnœa supervene, lasting sometimes for several days, or for a longer period, which are often most distressing. An attack of acute bronchitis is the most frequent cause of this paroxysm, especially in the first instance, although other causes which irritate the susceptible bronchi may induce it; as also, perhaps, remote irritations, as the influence of indigestion.

The cause of the dyspnœa, permanent as well as paroxysmal, is capable of a satisfactory explanation. The hypertrophy of the walls of the air-cells in which the capillary vessels of the lungs ramify, by increasing the thickness of the medium between the blood and air, is, of course, an impediment to the action of the air upon the blood, which is the primary object of respiration. Hence, an instinctive, increased effort on the part of the lungs to supply more air, and the dyspnœa, which is the result of the want and of the effort. In incipient cases, this want is only experienced when the current of the circulation is quickened by any cause, especially by active exercise. But in the progress of the case, as the hypertrophy of the cells increases, and more cells become implicated, the tendency to dyspnœa from slight causes, and even without an apparent exciting cause, becomes more marked. But in addition to this hypertrophy of the cell-walls, these walls are gradually absorbed by pressure, and with them the capillary vessels which ramify upon them. So that in time, a positive alteration takes place in the capillary circulation of the lungs. The thickness of the hypertrophied cell-walls not only interferes with the action of the air on the blood, but the amount of blood circulating through the pulmonary capillaries is diminished. Finally, the pressure upon the capillary vessels by the hypertrophied air-cells crowding upon each other, the actual destruction of many of these capillary vessels by absorption, produce a decided obstruction to the circulation of the blood through the lungs, which is felt first by the right side of the heart, and subsequently by other portions of the organ, thus leading to enlargement of the heart.

These causes united, render dyspnœa an early, constant, and a progressively increasing symptom in emphysema.

But the dyspnœa is frequently, especially in advanced cases of the disease, liable to be aggravated by paroxysms. These paroxysms are not directly connected with the condition of the air-cells, for they may occur without emphysema. They are dependent upon that peculiar irritability, or tendency to spasm, which this disease in its progress seems to impart to the bronchi. Under the influence of irritation, inflammatory or otherwise, they contract, and refuse to admit fresh air, or to discharge that which has already been used by the lungs. Thus the permanent but endurable dyspnœa of emphysema becomes aggravated to an alarming oppression and want of breath.

This spasm of the bronchi plays a most important part in the paroxysms of emphysema, for when this is overcome, the dyspnœa subsides to its usual degree, and the relief is often so great that the patient hardly complains at all of his permanent oppression. There can be no doubt that bronchial inflammation, sometimes severe, but often very moderate in degree, is the principal exciting cause of the paroxysm. So susceptible do patients sometimes become to this influence, that a very moderate exposure to cold and dampness, or even a mere change in the weather, without evident exposure, will irritate the bronchi to spasm, and induce a paroxysm. No doubt, in many cases, the constitutional tendency of the patient, or the peculiar condition of the system at the time, will increase the irritability of the bronchi, and induce a paroxysm without the necessity of any inflammatory excitement. Thus, the inhalation of certain odors will sometimes excite a sudden paroxysm of dyspnœa. I knew a medical gentleman in this city, long subject to pulmonary irritation, and in his youth supposed to be tuberculous, but whose breathing was usually free from dyspnœa, who could never uncork a bottle of ipecac to administer a dose to a patient. The odor at once induced a paroxysm of dyspnœa, which, in one instance at least, continued with great severity for three days, and finally terminated in copious mucous expectoration. Other persons are similarly affected by the odor of new hay. Indigestion, connected with an irritable condition of the mu-

cous membrane of the stomach, is also capable of increasing the permanent dyspnœa, as well as of inducing a paroxysm. A patient may suffer from indigestion, and the chief symptom be a considerable degree of dyspnœa, and that without any marked evidences of gastric derangement, until the case is carefully examined, and the effect of treatment noticed. Finally, mental emotion, in persons of a nervous temperament, may induce a paroxysm.

This dyspnœa, permanent and paroxysmal, is the most characteristic symptom of emphysema. Yet it cannot, as I shall explain to you as I proceed, be regarded alone as diagnostic of the disease.

A patient affected with emphysema will perhaps tell you that his breathing has been "short" ever since he can remember it, but that this did not annoy him until of late years; or, if this symptom did not exist in early life, it commenced at a later period, and increased very gradually, giving him very little trouble, so that he could continue his usual occupation, which is perhaps rather laborious. He did not emaciate, or lose strength, his appetite and digestion continued good, he was free from fever—in a word, the vital functions, except the respiration, were all well enough performed. By-and-by, he began to cough, and to experience occasional palpitation, at first, perhaps, with very little inconvenience, but at length, after exposure, he contracted a severe bronchitis, and a paroxysm of intense dyspnœa: a dry, fatiguing cough, wheezing respiration and febrile excitement speedily followed.

The aggravated symptoms of the paroxysm seldom continue longer than one or two weeks. As the expectoration becomes more free, the dyspnœa abates, and the respiration returns to nearly its former condition. I say nearly, for if the paroxysm has been pretty severe, I am inclined to think that the permanent dyspnœa will be found to be somewhat increased, and the cough to have become a rather more prominent symptom. Sometimes, however, the cough subsides entirely after the paroxysm, especially when it did not previously exist, as may have been the case.

During the paroxysm, the patient will lose some flesh and

strength, and his digestive organs will be more or less impaired, but these constitutional symptoms soon subside, unless the permanent dyspnœa is very considerable.

These paroxysms continue to recur, and from slighter causes. They thus increase in frequency, and from the gradual increase of the disease they become also more violent, increasing the permanent dyspnœa, and with it the constitution at length suffers. The countenance becomes permanently pale and bloated, with a bluish tint, and the eyes project; there is also a tendency to œdema of the lids; the strength is impaired, and some degree of emaciation ensues; palpitation becomes a more prominent and a more permanent symptom; and finally, the feet begin to swell from œdema; at first, during the paroxysms, afterwards, during the intervals also. These symptoms, indicating that the heart is seriously implicated, indicate also that the final issue of the case is approaching. The patient dies, exhausted by the increasing oppression, and with dropsical symptoms.

During a paroxysm of dyspnœa, or of asthma dependent on emphysema, and induced by an attack of acute bronchitis, you will find the patient sitting up in bed, or in a chair, often with the windows open. The face is pale, the lips often livid, the skin a little warmer than natural, the pulse somewhat accelerated. The respiration is laborious, often exceedingly so, calling into action the muscles of the neck, and accompanied by a wheezing sound. Every now and then, there is a noisy, dry, fatiguing cough, which brings no relief, but on the contrary, increases the distress. There is a sensation of stricture across the chest, and the distressing sensation of a want of air. The patient has but little inclination for food, and is restless, and disturbed in his attempts to sleep. If you examine the chest under these circumstances, you will find it everywhere resonant on percussion, probably unusually resonant, at least in some portions, while the respiratory murmur, feeble and indistinct in portions of the chest, harsh and dry in other portions, is almost everywhere masked by a sibilant, or by a sonorous rhonchus. In a few days the paroxysm gradually subsides, and at the same time the slight febrile symptoms disappear. The appetite improves, the expectoration becomes more free, and the cough

more easy; a greenish muco-purulent expectoration gradually becomes established, and with this, often a mucous rattle over the posterior and inferior portions of the lungs, while the sibilant and sonorous rhonchi disappear, or materially diminish—lingering in those portions of the lung most affected.

If you examine the patient during the interval between the paroxysms, you will have a better opportunity to observe the leading physical signs of the disease. Let the patient's chest be fairly exposed, in an erect or in a sitting posture, and you will frequently observe, when the emphysema has become general, that the whole chest is remarkably full and rounded, both before and behind. But in other cases the disease is limited in extent, but more advanced in degree. Perhaps in a particular portion you will notice a distinct bulging of a portion of the chest, as over the anterior portion of the upper lobe of the lung. This prominence is readily seen to be at the expense of the intercostal muscles, as well as of the ribs. It may exist also above the clavicle, filling up the hollow space which naturally exists there. Sometimes this prominence exists over the precordial region, sometimes it is quite marked posteriorly, raising up the blade of the scapula to a considerable degree. It also happens, in well-marked cases of general emphysema, that the arch of the diaphragm is depressed, and with it the liver and other organs.

This increased development of the chest is accompanied by a decided increase of the sound on percussion. The whole chest may resound remarkably well, but over the dilated portions it is still more clear. With this increased resonance the respiratory murmur is feeble, and sometimes almost extinct. Occasionally this is masked by a sibilant rhonchus, which disappears after coughing.

In certain cases, aggravated cases, instead of the feeble respiratory murmur over the chief seat of the disease, you will perceive a distinct, but harsh, unexpansive sound. It accompanies equally the effort of inspiration and of expiration. Its mechanism has never been satisfactorily explained. Does it come from the smaller bronchi, through the rigid and inactive pulmonary substance? Has it its seat in the large and broken-

up air-cells, which have passed beyond the state of simple hypertrophy and dilatation into the honeycomb condition, produced by many cells united to form a few large ones?

Dilatation of the parietes, increased resonance on percussion, feeble or harsh respiratory sounds, are the characteristic physical signs of pulmonary emphysema. I have mentioned them in the inverse order in which they develop themselves. Thus, in an incipient case, you will perceive a feeble respiratory murmur, united with a clear sound on percussion. At first these signs may be doubtful, but gradually they become more developed, and with this, the enlargement of the parietes occurs to confirm the diagnosis.

I have mentioned a cough as a prominent symptom in emphysema. It often is so, but not always. It is not essential to every stage of the disease. In the early stage, especially in congenital cases, it is often absent. When it does appear, as a symptom of a concomitant bronchitis, it may cease when the paroxysm ceases, or continue in so slight a degree as hardly to be noticed, especially during the warm season of the year. In time, however, it gradually becomes more permanent, as well as more prominent, and but few cases of well-developed emphysema are without it. The expectoration may at first be trifling or entirely absent, gradually it becomes more abundant and frothy, and assumes a dirty hue, like a solution of gum-arabic. When the bronchitis becomes acute, this expectoration ceases for the most part, giving place, first to the frothy transparent mucus, then to the opake, greenish, muco-purulent expectoration of ordinary bronchitis—to resume its former character when the acute attack has passed.

Pain in the chest is not a striking symptom in emphysema. There is frequently a sense of stricture, or a feeling of oppression, or of general uneasiness in the chest—to which patients become accustomed by long habit, so as seldom to complain of it.

When, in the course of the disease, you find that palpitation, which has, perhaps, for a long time been occasional, and moderate in degree, becomes more constant and troublesome, and especially when you notice œdema of the lower extremities,

occurring at first only during the paroxysms, but tending to become more permanent, you should infer that enlargement of the heart has taken place. A careful examination of the precordial region will often reveal distinctly the physical signs of this condition. The apex is displaced, the impulse is extended, while the sounds of the heart usually remain natural, and the action moderate. Enlargement of the heart, in these cases, is rather a passive than an active change, and consists of dilatation of the organ rather than of hypertrophy, although both conditions are usually found united. The cardiac disease probably commences in the right side, and extends finally to the whole organ. In cases of enlargement of the heart connected with emphysema, you must not be deceived if you find the sound on percussion even clear, over the precordial region, for the lung is often emphysematous in this situation. Neither must you necessarily attribute a bulging in this region to the condition of the heart, as it may occur from emphysema also, and present precisely the same characters in both diseases. Precordial prominence, with the characteristic mark of internal pressure—that is, equal dilatation of the ribs and of the intercostal spaces—if united with dulness on percussion, is probably dependent on an enlargement of the heart; if, however, it is united with unusual clearness on percussion, it is probably connected with emphysema.

In the description I have given of a paroxysm of emphysema, I have referred only to one cause, acute bronchitis, the most common cause of the paroxysm. But other irritating causes, acting on irritable bronchi, will produce the same result. Thus, there are some persons in whom the inhalation of certain odors, as that of ipecac, of new hay, will induce a paroxysm, while others are sure to be affected by visiting certain localities without your being able to ascertain any good reason for the effect produced. Generally speaking, emphysematous persons breathe badly in dry and elevated regions, or when the atmosphere is damp and chilly; and, finally, attacks of indigestion may lead to the same result. In persons of a nervous temperament, as well as in those who have long suffered from the disease, very trifling causes will induce spasm, and thus aggravate very much the

dyspnœa. Generally, however, the attack is of less duration than when induced by inflammation of the bronchi, although it may continue several days, and finally terminate with free expectoration.

It is possible that this tendency to spasm of the bronchi may be the only difficulty, and thus, under the influence of irritating causes, paroxysms of dyspnœa may be induced without the existence of emphysema. A *nervous* or *spasmodic asthma* may, in fact, exist without any organic lesion of the lungs, discoverable by the most careful post-mortem examination. It is easy to conceive that a general spasm of the bronchi should induce a great degree of dyspnœa, and you should also, in such a case, expect that when the spasm subsided, the breathing would become quite free and natural. So, indeed, it may, if the spasm entirely subsides; but if it continues in a certain degree, as is very likely, then a slight degree of dyspnœa will continue with it. Permanent dyspnœa, however, belongs to an organic affection, like emphysema, rather than to a purely nervous affection.

In the nervous variety of asthma, you can often gain important assistance from the physical condition of the chest. You will find there none of the prominent signs of emphysema—dilatation, increased resonance on percussion, feeble respiration. But you may hear the sibilant and the sonorous rhonchi, especially during a paroxysm, for bronchial spasm will readily produce these sounds.

It is not very uncommon to meet with individuals who are annoyed by a paroxysmal dyspnœa in certain localities, who, if their statement can be believed, breathe perfectly well in other localities. If this fact can be perfectly established in any case, there will be reason to think that the affection is purely nervous. But you should be careful in your examination of such cases. What patients call perfect relief, is sometimes found on examination to be but partial relief. If a slight dyspnœa remains, you should suspect the existence of emphysema, although possibly this symptom may be, after all, owing to a slight continuance of the spasm. You should carefully examine the chest.

Children are sometimes quite asthmatic, and they grow out of it, as the expression is. In these cases emphysema can hardly

have existed, for there is no evidence that it ever disappears, when it has once existed. It, on the contrary, tends slowly to increase.

The existence of a purely nervous asthma, presenting many of the symptoms of emphysema, must, I think, be admitted. It is not, however, a common affection.

There is another affection presenting many of the symptoms of emphysema, to which I wish to call your attention. It may, in this connection, be properly enough called *cardiac asthma*. In organic disease of the heart, usually when the disease is well established, attacks of acute bronchitis are apt to supervene, inducing sudden and distressing dyspnœa, and resembling very much, at first sight, a paroxysm of emphysema from bronchitis. But a proper inquiry will satisfy you that the heart symptoms have long preceded the attack, and a physical examination of the chest, while it reveals, it is true, the evidences of a general bronchitis with spasm—an extended sonorous or sibilant rhonchus, a mucous rattle often abundant—yet none of the permanent signs of emphysema are found to exist; no dilatation, no increased resonance. It is a fact, however, that disease of the heart does induce a condition of the bronchial tubes quite analogous to that induced by emphysema, viz., a tendency to inflammation, and particularly to spasm; a tendency which is felt to be particularly unfortunate for the patient, when the two diseases exist together.

It is not uncommon for tubercles in the lungs to be developed during the progress of emphysema. If the patient emaciates progressively, if the pulse is permanently accelerated, if hectic supervenes, and especially if hæmoptysis occur, you may reasonably suspect the existence of tubercles in the lungs. Hæmoptysis is not a symptom of emphysema; it should always excite the suspicion of tubercles in the lungs. In these complicated cases of emphysema and of tubercles, the physical diagnosis is often puzzling, inasmuch as the organic changes in the two diseases are antagonistic. In emphysema the lung is dilated, rarified; in tubercles, it is contracted, condensed. And as the chief seat of the disease is the same in both diseases, viz., the anterior and superior portion of the lung, it is easy to estimate the difficul-

ties of the case. Emphysema has already induced a dilatation, an increased resonance on percussion, a feeble respiratory murmur under the clavicle. Tubercles, when they are deposited in the same situation, tend to diminish this dilatation, and especially the resonance, while they do not materially alter the character of the murmur, except perhaps in rendering it more feeble. So that it is easy to conceive a case, in which a considerable deposit of tubercles has taken place in an emphysematous lung, and yet in which the formation of the chest and the resonance on percussion shall have only receded to a degree which may be considered natural, while great feebleness of respiration remains, and probably with it, may be added unusual resonance of the voice, or broncophony. In a case of emphysema, therefore, where tubercles are suspected from the constitutional or the rational symptoms, and the respiration is simply feeble at the summit of the lung, but with increased vocal resonance, the existence of tubercles is probable.

What I have just stated, applies to the early stage of the tubercular deposit. When the tubercles begin to soften, a mucous rattle is developed at the summit of the lung, without existing elsewhere. This at once changes a probability into a certainty. This single sign is highly characteristic of tuberculous disease. It does not belong to emphysema under any circumstances.

In the more advanced stage of the tuberculous disease, when cavities have formed in the lung, the diagnosis becomes still more clear.

Slight cases of emphysema occur which have been mistaken for tuberculous disease of the lungs, even by those tolerably experienced in auscultation and percussion. A young person, perhaps, of rather delicate constitution, notices habitually a slight degree of dyspnœa, and a trifling cough. The chest is examined, and a slight degree of dulness on percussion is detected beneath one of the clavicles. This may lead to the suspicion of tubercles. But this dulness is apparent, not real. It exists by comparison only, because a slight degree of emphysema in the opposite lung has increased somewhat the natural resonance on percussion. If, in this case, a slight fulness of the walls of the chest should happen to exist about the clavicle, where the em-

physema is seated, then the opposite space might not only sound dull, but seem depressed; and thus a new suspicion of tubercles might arise. But a little attention to the respiratory murmur will correct these errors. Where the apparent dulness and depression exist, the respiratory murmur is *distinct and natural.* On the emphysematous side it is feeble. The constitutional symptoms of tubercles are, of course, absent in these cases.

The permanent dyspnœa, dependent upon organic changes in the air-cells and in the capillary circulation of the lungs, is not only an incurable affection, but incapable of being materially influenced by treatment. But the bronchial spasm, which aggravates the permanent dyspnœa, not only by inducing a paroxysm, but, more or less constantly, by continuing to exist in a less degree during the interval. In old cases of emphysema, you can seldom apply the ear to the chest without catching more or less of the sibilant or the sonorous rhonchus, especially in those portions most affected. This spasm must increase the permanent dyspnœa, and, being liable to aggravations from slight causes, the dyspnœa will be liable to variations in degree, all of which, however, fall short of a distinct paroxysm of asthma. When this irritability, this spasm of the bronchi, exists, you must try and learn what aggravates it. Is it indigestion? Is it some moral influence? Is it the peculiar state of the atmosphere in which the patient lives; and, especially, is it a trifling exposure to cold and moisture which aggravates this condition? If you can ascertain the existence of any such exciting cause, the indication is clear. Any irritation in the digestive organs must be removed. Low diet, leeching, the warm bath, may be necessary; and, after a time, the administration of tonics, of iron, of the mineral acids, of quinine may be of service. If an hysterical or nervous temperament exposes the patient to the influence of unfavorable moral influences, these must be avoided if possible. A change of residence, exercise, agreeable society, must act beneficially in such cases. If the lungs are extremely sensitive to cold and to other climatic influences—and this, I believe, is a very frequent cause of aggravation to the dyspnœa—a change of residence will often work wonders. The facts which illustrate this are very curious. An asthmatic patient, who breathes

with great difficulty for the most part of the time in the city, will find the greatest relief by residing in the country. Another patient will find the same benefit by moving from the country to the city, or from one city to another, or from one residence in the country to another. The distances necessary to produce these favorable results are often very trifling, and the differences in other external circumstances are quite inappreciable. A high, elevated region seldom agrees with emphysema; neither do damp and low situations. The sea is particularly beneficial to such patients, while a residence on the sea-coast is, usually, injurious.

Other circumstances are worthy of attention in controlling the permanent dyspnœa. A careful attention to diet by the use of simple unstimulating food; care against exposure to currents of air, and to evening dews; and especially the habit of cold sponging. I have long been in the habit of recommending sponging of the chest, with subsequent friction, to invalids with delicate lungs. Cold water may be used, if reaction is readily induced, or it may be warmed to suit the condition of the patient. This habit, faithfully continued every day (and the time of rising in the morning is the best time to practise it), often produces the best effects, by fortifying the chest against the influences of a bad climate. Care should also be taken to guard the person by proper clothing. Flannel should be worn next the skin during every season of the year. The general use of woollen clothing, even in summer, should be enjoined, and the invalid should never leave home for any length of time without being provided with clothing suitable to a sudden change in the weather, and, especially, with an outside garment. Invalids who find themselves thus sensitive to sudden changes are apt to grow nervous, and, perhaps, to take too many precautions. Their thoughts dwell constantly upon their condition, and this alone aggravates their sensitiveness. Those who enjoy the best health know very well, that when the mind is powerfully acted upon and the attention drawn from themselves, that great exposure is often risked with impunity. The influence of the mind in inducing and aggravating all affections into which a nervous or spasmodic element enters is well known; yet, if too much caution, and the habit of concentrating the attention upon a "weak spot" which

may happen to exist, is injurious, a reckless indifference is still worse. The emphysematous invalid cannot try the influence of careless exposure long, without being reminded of his folly by a paroxysm of his disease.

By care and prudence, by understanding what aggravates the disease, and by avoiding it, the comfort of the invalid may be much promoted, and the frequency and the violence of the paroxysms be diminished, and, probably, the progress of the disease be materially arrested.

When you are called to a patient suffering from a paroxysm of emphysema, you should first ascertain what has been the exciting cause of the paroxysm. In a great majority of cases, you will find this to be acute bronchitis. The indication is to remove or to diminish this bronchitis as soon as possible. Tartarized antimony in nauseating doses, or ipecac; opium, pediluvia, mustard poultices to the chest, diluent and mucilaginous drinks, are the best remedies to effect this result. The breathing becomes more easy, the patient begins to expectorate, the slight febrile excitement subsides, the sibilant rhonchus diminishes, and, in certain cases, a mucous rattle begins to be heard at the base of the lungs. If the patient be very plethoric, and not advanced in life, and the paroxysm an early one and very severe, it may be advisable to practise venesection. In such cases it will produce prompt and decided relief. But it is a remedy that should be used only in cases in which it is strongly indicated, and it should never be relied upon to mitigate the paroxysms, as they return from time to time. Indeed, if bleeding be repeated in these cases, it seems to lose its immediate good effect; and its influence on the constitution and future health of the patient is by no means favorable. If the patient be gouty, and gout is not a rare complication in emphysema, colchicum is a most valuable remedy. It may be given, if the bronchitis is severe, in combination with tartarized antimony, and afterwards with the carbonate of potassa. This latter combination frequently acts as a diuretic, and this adds to its favorable influence.

In many cases, when the inflammation of the bronchi is moderate, and the spasm more prominent, alkaline remedies exert a favorable influence, and may very properly be combined with

other agents in the treatment of the paroxysm. Thus, they may be combined with stimulating expectorants, in many cases, when the paroxysm is declining.

In advanced cases of emphysema, and especially in nervous, irritable constitutions, the spasm is the chief element in producing the paroxysm. There may even be no bronchial inflammation to excite it. Such attacks are usually sudden in their development, and rapid, as well as variable in their disappearance. There is no febrile excitement present; but often a coldness of the surface, and especially of the extremities, with cramps sometimes. In this condition of the system, antispasmodics are indicated. Opium possesses great power over these paroxysms. Some patients experience great relief from smoking stramonium in a common pipe. I have known the inhalation of the fumes of nitre, prepared by dipping paper into a saturated solution of nitre, and, after drying it, burning it in the room, to be attended with the greatest relief; while, in other cases, it has failed entirely. You should be careful in these spasmodic paroxysms to inquire carefully for the exciting cause. Perhaps it may be indigestion, and the dyspnœa will subside as the stomach is relieved; perhaps it is caused by mental emotion, when it may be followed by a paroxysm of hysteria; perhaps the patient has inhaled some irritating substance, as ipecac, the effects of which are not as transient as might be imagined. I have known a paroxysm from this cause continue three days, and then to subside with a copious mucous expectoration, as if bronchitis had supervened upon the spasm.

Other antispasmodics have been much employed in this disease to relieve the paroxysm. Lobelia has been recommended; but this is an uncertain and somewhat dangerous remedy. Tobacco has also been used for the same purpose, and strong coffee exerts a favorable influence in many cases.

But when the spasm has become predominant, and the patient suffers from very slight exposure to exciting causes, he has still a resource in a change of climate, and especially in a sea-voyage. The influence of this change is sometimes very great; but the patient must continue this course, if he expects permanent relief. If he returns home, his paroxysms return also, and as severely

as before. I remember the case of a gentleman, a native of Belgium, who suffered, almost constantly, while in this city, who was almost entirely relieved during a residence in Europe for a considerable period of time, but as soon as he returned, he suffered as severely as ever.

Preparations of iron and other tonics have sometimes been found to exert a favorable influence in controlling the spasm; but I am inclined to believe that their influence is very moderate. The best course for the patient to pursue, is to watch carefully, and observe what excites this spasmodic action, and to avoid it as far as possible.

An attack, especially if induced by exposure to cold, may sometimes be much mitigated by proper attention. When the patient begins to complain of chilliness, with lassitude, and a commencing stricture of the chest, let him at once retire to bed, let him have a mustard pediluvium, let him take a diaphoretic mixture, and remain quiet. The paroxysm may pass away so lightly as hardly to give him any discomfort.

When symptoms of enlargement of the heart ensue, and dropsical effusions add to the distress of the patient, the chances of relief are much diminished. You must, however, try to control the effusion, and to diminish it, if possible, by diuretic remedies, by purgatives, and by other appropriate means. Such patients are usually advanced in life, and enfeebled by long disease. It is advisable, therefore, to combine a tonic treatment with other means. A combination of the acetate of iron with the acetate of potassa, I can recommend as a suitable prescription in such cases.

I have stated, that paroxysms of dyspnœa, and perhaps a certain degree of permanent oppression, may occur without evidence of emphysema or of other organic change. In these cases, an irritable condition of the bronchi is the only prominent feature. Time, and the changes of life, will sometimes overcome this tendency, especially when it occurs in children. Tonics, cold sponging, a regulated diet, may also be useful. But when other means fail, a change of residence should always be recommended. It is very remarkable sometimes, as I have already stated, how a slight change will be followed by the most beneficial results.

LECTURE XIV.

TUBERCLES IN THE LUNGS.

Pathological anatomy.—Stages of the disease.—Indications of a curative effort.

Tuberculous phthisis is the most frequent as well as the most fatal disease of the lungs that the practitioner of medicine is called upon to encounter. It is, therefore, a subject of great interest. Every thing connected with its pathology, its development, its progress, and its termination should be well understood.

Tubercles in the lungs present three distinct stages. They are commonly first deposited in the form of small granulations, varying from the size of a pin's head to that of a small pea. They assume, more or less, the round form, but imperfectly; indeed, they are often angular. They are grayish, semi-transparent, and feel firm when pressed by the finger. In this stage they are called miliary tubercles.

After a time,* these granulations assume a yellowish-white aspect, increasing at the same time in size. A yellowish spec commences in their centre, sometimes towards their circumference, which gradually extends until it gains possession of the whole granulation; which now, having become yellow and opake, and increased in size, is called the crude tubercle.

Finally, the yellow or crude tubercle begins to soften in its centre, and this softening gradually extends to the whole mass, until a small cavity is formed, containing a thick, yellow, pasty, or purulent-looking fluid, which, however, in its simple state, does not contain a single pus globule.†

The tuberculous deposit in the lungs does not always take

* The crude, yellow tubercle, according to Lebert, is not unfrequently the primary deposit, and does not differ from the gray granulation, in the character of its ultimate microscopic elements, but only in their proportions.—See Appendix.

† For the microscopic appearances observed during the period of softening, see Appendix.

place in the form of distinct granulations. Sometimes it is infiltrated into the pulmonary tissues, even in large masses. But this is only a difference in form. The grayish semi-transparent infiltration is but the miliary tubercle in another form. After a time, numerous yellowish points are developed in its substance, which increase and become confluent, until the whole mass becomes yellow and opake, like the crude tubercle. Finally, the mass begins to soften at different points, and to break down into a thick, yellowish, opake detritus. In children it is frequently deposited in patches of considerable size, immediately beneath the pleura covering the lungs.

The seat of the tuberculous deposit seems to be, generally, the cellular tissue of the lungs. It may be seated in the air-cells, or in the bronchi, it may even be effused upon the free surface of the mucous membrane of the lungs. But its most frequent seat in these, as well as in other organs, is the cellular tissue.

The first effect of a tuberculous deposit is to compress the surrounding tissues. Experiments seem to prove that this compression renders the lung, in the immediate neighborhood of the deposit, less vascular than natural, the small capillary vessels being obliterated. But, after a time, the capillary vessels a little more remote become enlarged, and, perhaps, increased in number, so that, finally, a state of hyperemia may exist in the portion of lung affected.

It is difficult to say, exactly, what the condition of the lung has been during life, by observing the appearances which it presents after death; and this is especially true of the vascularity of the organ. But this fact is quite well established; that when you examine the lungs which are the seat of the tuberculous deposit in its early stage, you will commonly find them pale, free from vascularity, and the tissues somewhat compressed by the deposit in its substance.

But this absence of vascularity is not permanent. Sooner or later, especially when the tuberculous deposit has become yellow and opake, and still more so when it begins to soften, evidences of increased vascularity become very apparent. More than this commonly soon occurs. Inflammatory changes will be noticed, which will go on increasing until the original appear-

ance of the part affected is entirely changed. Instead of the simple tuberculous deposit, and the pale and compressed pulmonary tissues, you will find congestion, ulceration, suppuration, and effusion of lymph.

These inflammatory changes are all secondary. The opinion is held by some pathologists, that a tubercle may form, increase, and even soften, without being attended by any inflammatory changes. But if this be true, it very rarely happens. Sooner or later inflammation supervenes, and constitutes a marked and most important feature of the disease.

The inflammatory changes which occur around tuberculous deposits are congestion, with effusion of lymph and of serum, but more particularly ulceration and suppuration. It would seem that when a tubercle begins to soften, inflammation takes place in the pulmonary tissues around it. These tissues—cellular tissue, air-cells, bronchi—ulcerate, and thus form a cavity around the softening mass. Suppuration takes place in this cavity, which, mixing with the tuberculous matter, forms a tuberculous abscess often communicating with a bronchus.

This tuberculous abscess is seldom formed about a single tubercle, although this is sometimes observed. Many tubercles clustered together prevent each little mass from possessing its own distinct inflammatory circle. They break down into a common mass of detritus, and thus form an abscess much larger and more irregular in shape than that formed by a single tubercle, which is rounded and small, like a pea-issue.

The abscesses thus formed by the combined influence of softened tuberculous matter and inflammation, present different appearances in different cases. They vary very much in size, being sometimes formed about a single tubercle, when they are not larger than a pea, but usually they are much larger, being sometimes capable of containing a pigeon's egg, a hen's egg, and even occupying nearly a whole lobe of the lung. They are often very irregular in shape, from the successive rupture of softened tuberculous masses into their cavities, and frequently present bands stretching across in different directions, which are the obliterated blood-vessels of the lung, less prone to ulceration than the surrounding tissues. The walls of these abscesses,

when recent, are bare, and formed of pulmonary tissue condensed by inflammation, and by the deposit of tuberculous matter. Sometimes the cavity is lined, but imperfectly, by a layer of unorganized lymph.

In the progress of the disease, if the abscess, gradually extending its limits, finally reaches a portion of lung comparatively free from the tuberculous deposit, an organized false membrane begins to form, at first imperfectly, and in patches, until at length it may form a more or less complete lining membrane to the abscess. This lining membrane, at least before its complete development, is still covered by layers of imperfectly organized lymph; but when its organization is fully completed, it forms alone the parietes of the abscess, which may in this condition be considered as fairly in the way of cicatrization. This lining membrane, at first thin and delicate, becomes gradually more thick and fibrous in its character. It may attain several lines in thickness, and become so firm as to resemble fibro-cartilage, although no true cartilage exists in its structure; it is simply fibrous. In certain rare cases, I have seen this lining membrane retain its original delicacy of structure; indeed, it appeared to be converted into a tissue very like a mucous membrane.

These abscesses, especially when of considerable size, communicate by ulceration with the bronchi; sometimes very freely, sometimes not so. Indeed, I have seen abscesses of very considerable size in which such a communication did not exist. I think that these cases are not infrequent. This fact is of some importance in the physical diagnosis of these abscesses, as I shall explain to you in a future lecture.

The contents of a tuberculous abscess are various. An ordinary glance at their contents would induce you to say that they contained pus. But a microscopic examination of their contents proves that they contain much more than this; viz., softened tuberculous matter, pulmonary detritus, epithelial scales, and sometimes globules of blood and of fat. This is the character of their contents while the process of destruction is still going on. But after the abscess becomes lined throughout by an organized false membrane, and the tuberculous matter has been

all expelled by expectoration, the secretion is purulent in its character, and this finally becomes changed into a serous, although viscid fluid, which may disappear in its turn if the cavity of the abscess becomes completely obliterated by cicatrization.

The cicatrization of a tuberculous abscess is not a very rare occurrence. It probably happens only when the deposit is limited in extent. But you will occasionally find, at the summit of a lung, a strong and old adhesion of the pleura, sometimes a crust of fibro-cartilaginous deposit, sometimes a band of considerable length passing from the lung to the ribs. In its neighborhood the lung is puckered, and sometimes drawn inwards by a deep linear depression. If you press it with the fingers it feels firm, consolidated, and to the eye it looks dark, from an abundant deposit of black pigment. If you cut into this portion, you will find, sometimes, a cavity lined by a grayish fibrous membrane, semi-transparent; or thick, whitish and fibro-cartilaginous; or soft and pliable like a mucous membrane. This cavity is usually small, capable of holding a pea, a cherry, or perhaps a plum, and it terminates abruptly in an open bronchus or a bronchus obliterated by a conversion into fibrous or fibro-cartilaginous substance. It sometimes contains a viscid, transparent, or reddish fluid, sometimes tuberculous matter which has undergone the cretaceous transformation, and which I shall presently describe. The surrounding pulmonary tissue is dark and solidified, and contracted by chronic inflammation. In other cases the cavity, which undoubtedly once existed, is obliterated, and in its place you will find a fibrous, or fibro-cartilaginous, or cellular solid mass, in which the bronchi abruptly terminate. This process of obliteration is sometimes noticed in its progress. The two opposing surfaces at the extremities of the mass are united to form a solid tissue, while a small central cavity still remains. In these cases of obliteration of the cavity, the surrounding lung presents the same appearances as when the cavity exists.

But there is another change much more frequently noticed, also at the summit of the lungs, which must equally be regarded as a curative process. You will find the same old adhesions at the summit of the lung which I have just described, the same

appearance of the pulmonary tissue, the same puckering and depressions, the same induration. But when you cut open the part you will find no cavity, no cicatrix, but one or more masses imbedded in the pulmonary tissues, usually of small size, the size of a pea or much smaller, rarely as large as a cherry-stone, which when examined look like portions of moist chalk. They are smooth and rounded, they break up under moderate pressure, and have a gritty, earthy feel. Sometimes you will find much harder masses, of a stony hardness, rough and irregular in shape, and usually smaller in size than the chalky concretions. You will find these masses closely attached to the surrounding pulmonary tissue, which is dark, contracted, and more or less indurated in their neighborhood. Sometimes the masses are inclosed in a distinct cyst. Indeed, they are found in cavities which are far advanced towards cicatrization, lying loose in these cavities, and surrounded by the fibrous or fibro-cartilaginous tissue which forms the walls of these cavities.

There is no doubt that these chalky masses, as well as the more firm masses, which are falsely called ossifications, are, in fact, successive changes of the same original substance, and that substance is tubercle. There is no doubt, also, that these changes follow the usual course of tuberculous changes, by beginning in the centre. Thus it has been noticed, that the outside of the mass will present the appearances of the yellow, cheesy tubercle; in a deeper portion, the chalky substance will be found; while quite in the centre, a small, irregular mass of stony hardness will be perceived. Not unfrequently, indeed, the chalky concretions are found united with the harder, irregular bodies; the two existing together in the same mass, but then the harder portion is always found in the centre.

The concretions are seldom numerous, and are always found at the summit of the lung. Their tuberculous nature is undoubted, but their small number and small size prove that the original deposit, of which they are the result, could not have been large. Every thing connected with their history proves that they exhibit one of the methods which nature employs to cure the disease, and that they may remain latent in the lungs, or create very little irritation during a long life. They are,

probably, never entirely absorbed, but gradually pass into the little stony, irregular concretions, which may be regarded as their ultimate stage. Neither does the surrounding lung, either in this case, or in the case of cicatrization, ever regain its natural condition. It remains, more or less, indurated, or contracted, and more or less impervious to air.

These cicatrices, or these concretions, one or both of them, have sometimes been found in the upper portion of the lungs, without any more recent tuberculous deposit. Except the small portion thus affected, the lungs are free from disease. Yet you will not unfrequently find that evidences of a more recent deposit of tubercles exist. Sometimes, indeed, they are numerous, and present all the stages of advancing phthisis. These facts must convince you that the cure of one crop of tubercles does not necessarily prevent the deposit of a new crop, and even the fatal termination of the case as phthisis. Still I think that you may safely infer, that the more advanced the progress of the curative process, in the first instance, the less likely is a new deposit to take place.

The formation of these concretions is found to be effected by the gradual deposit of mineral matter; the chloride of sodium, the sulphate of soda, united with a little phosphate and carbonate of lime, and sometimes with cholesterine, in the place of the proper matter of tubercle.

It would not be correct to say, that every time a fibrous or a cellular mass is found in the pulmonary substance, even at the summit of the lung, it is a proof that a tuberculous cicatrix exists. If no connection with a bronchus existed, if no chalky concretions or other tuberculous deposit was noticed, the fact would be more than doubtful. It is undoubtedly true that these fibrous, fibro-cartilaginous, or cellular masses, are not unfrequently found at the summit of the lungs, without having any thing to do with the disease in question. Masses of this kind are generally superficial, and seated in the sub-pleural cellular tissue, while the true cicatrix is found deeper in the substance of the lung. These sub-pleural formations are undoubtedly the result of a limited inflammatory action; they are the consequences of a deposit of lymph in the first instance, which subse-

quently undergoes its usual transformations. Adhesions of the pleura, and puckering, and slight condensation of the surrounding lung, frequently exist with them.

Again: every little stone-like or bone-like concretion that is formed in the lungs is not necessarily a converted tubercle. This condition may result also from a small deposit of lymph which passes gradually into a cartilage-like or bone-like condition, or it may be owing, as some think, to a partial ossification and obliteration of the smaller bronchial tubes. You must judge of these cases only after a careful examination.

I am not aware of any other methods which nature adopts to cure tuberculous disease. Can miliary tubercles be absorbed? This question has never been answered satisfactorily. They sometimes present a shrivelled appearance, as if they were disappearing from the lung, but I know of no facts which prove that they are ever absorbed, and that the lung ever regains its former natural condition.

The tuberculous deposit affects, by choice, the superior portion of the lung. The miliary tubercle has a tendency to cluster in distinct masses, although there are many striking exceptions to this rule. You will not unfrequently find the tuberculous bodies disseminated through the pulmonary tissues.

The *tuberculous infiltration* is apt to attack a large surface of the lung, and, contrary to the general rule in the miliary form, it not unfrequently attacks, in the first instance, the *lower lobe* of the lung. The tendency of the tuberculous deposit to affect, primarily, the superior portion of the lung, is a general law of great value, especially in the physical diagnosis of this disease, and the exceptions to it are so rare, that setting aside a few cases of primary tuberculous infiltration, they hardly need be considered. But while the tuberculous deposit almost always first affects the superior portion of the lung considered as a whole, it is by no means unusual to find the deposit commencing at some distance below the apex, and leaving that portion of the lung entirely free from disease. This fact should also be remembered in the physical diagnosis of phthisis.

The tuberculous deposit possesses also another peculiarity of great importance. It takes place in successive crops, so to speak.

The first deposit is usually limited in quantity and extent. It is probably not more than the efforts of nature could get rid of without endangering life. But while the first deposit is advancing in its stages, a new deposit takes place, and then another, until at length the lungs become too far disorganized to admit of cure. It is probable, also, that in the first instance the disease is often, perhaps generally, confined to one lung; but, as successive deposits take place, both lungs become affected. This view of the case will serve to explain what might otherwise puzzle you in your post-mortem examinations. You will find, indeed, cavities, crude tubercles, miliary tubercles, tuberculous infiltration, all existing together in the same portion of the lung. But their appearance is the evidence of successive deposits: the earlier deposit has formed cavities, that of the middle period of the disease exists as crude, yellow tubercles, while that of recent formation presents the miliary, the grayish semi-transparent appearance.

Still, when you look at a tuberculous lung as a whole, it will be evident, at least in a vast majority of instances, that the disease has spent its chief violence upon the superior lobe. It is there that you will find the oldest and the largest cavities; it is there, in cases of cure, and there only, that you will find cicatrices and the cretaceous masses.

As a general rule, there is no difficulty in distinguishing the existence of tuberculous disease in the lungs, even when the disease has been limited in extent, and has been cured, as far as it can be, by the efforts of nature. A tuberculous cicatrix does not necessarily differ from any other cicatrix, so far as I know. But a cicatrix from any other cause is so rare that you can seldom have the means of comparison. But generally, independent of the seat of the cicatrix near the summit of the lung, you will commonly find a few chalky concretions, or shrivelled miliary tubercles, to indicate more distinctly the true character of the case. The chalky concretions also, although very unlike tuberculous matter in appearance, are yet characteristic, and are often mixed with more or less dry and friable tuberculous matter. They may, however, be confounded with certain bone-like deposits found in the lungs, sometimes as small particles, sometimes

in masses of considerable size, and which are not all tuberculous in their nature. They are the consequences of effused lymph, which, instead of being absorbed, has been gradually converted into this bone-like substance.

In certain rare cases, you will find in the lungs, I think that I have seen the appearance twice, a deposit of small, oval, flattened, yellowish semi-transparent little bodies, all of the same size, and disseminated equally through the lungs, which might, at first sight, be supposed to be miliary tubercles; but their uniform size and their equal dissemination, their shape also, will readily distinguish them. They were described by Bayle, and called by him accidental cartilages. Broussais ingeniously supposes them to be enlarged lymphatic glands. Their true nature and progress are unknown.

The precise nature of the tuberculous deposit has not yet been ascertained. It may be regarded as one of the manifestations of the scrofulous diathesis. But in making this statement, I offer no explanation of its real nature. It has been regarded by some pathologists as imperfectly developed fibrine, incapable of organization, and the result of defective nutrition. It is supposed that the albuminous matter of the food, as it passes into the circulation, becomes more highly vitalized by contact with the living tissues and becomes converted into fibrine, and that the healthy nutrition of the different tissues is thus supplied. But when this vitalizing and converting process is imperfect, it is thought that a portion of the albumen or imperfectly developed fibrine is secreted into the tissues of the lungs and of other organs in the form of tubercle. This doctrine is, however, at best, but an ingenious hypothesis. Animal chemistry has not as yet been able to reveal the true nature of the tuberculous deposit. The best analysis is that of Preuss. It teaches that tubercle contains a great many ingredients in its composition, of which casein is one of the most important.

The microscope has succeeded better than chemistry in the minute investigation of the tuberculous deposit. The best observers have discovered a peculiar cell, which enters largely into the formation of tubercle, and which they regard as characteristic of this deposit. It is observed in the miliary tubercle,

but less perfectly developed, and mixed with other elementary matters. Most perfectly seen in the crude, yellow tubercle, it becomes disintegrated and disappears, when the tuberculous matter has softened, so that, unfortunately, it is not often detected in the expectoration. It would be useless to attempt to describe this peculiar tubercle-cell, which I have often examined with the microscope. I prefer to exhibit it to you, so that you may learn to judge accurately of its appearance, as well as learn the mode of preparing it for microscopic examination. (See Appendix.)

If the tubercle-cell is recognized as a distinct form of elementary cell, it establishes the fact, long since contended for by pathologists, that tubercle is a heterologous formation—that is to say, different from any of the normal tissues of the body, thus constituting one of an important class of diseases, of which class cancer is another striking illustration.

It has been a question whether tubercle was a vitalized product or not. It is very certain that blood-vessels do not exist in its substance, at least as a general rule. I have seen a single specimen, prepared in London, which is intended to represent a capillary vessel passing into a tubercle. Lebert, in his microscopic observations, remarks, that he has observed the same thing, perhaps once or twice. But is it not more probable that in these rare and exceptional cases, the tuberculous matter has been deposited around the capillary vessel without obliterating it, rather than that the vessel itself had entered into the tuberculous structure?

The changes that are observed in the tubercle, commencing generally in its centre, have been regarded as a proof of at least an imperfect vitality, and this is, I think, a correct opinion. The change from the semi-transparent miliary tubercle to the opake, crude tubercle, is now explained by the microscope as consisting chiefly in an accumulation and increase of the tubercle-cells, commencing in the centre, and extending to the circumference. But this accumulation implies vitality, but not necessarily the presence of vessels—one cell secreting other cells.*

* The theory of Schwann, that a cell is the primary element of all organized tissues, has been looked upon with much favor by physiologists. It has also been assumed that heterologous formations, among which is tubercle, are composed of

It is also a question whether the secretion of the tuberculous matter is uniform; that is, whether it always first assumes the characters of the gray semi-transparent tubercle, or whether it may not also be deposited, *ab origine*, as the yellow, opake tubercle. If the chief difference between the miliary and the crude, yellow, opake tubercle is a difference in the number and in development of the primitive cells, it is easy to conceive that this may happen; and do facts seem to warrant this belief? Is the yellow, opake tubercle found as a primary deposit? I think not, unless in exceptional cases.

The true appearance of recently deposited tubercle is that of a whitish semi-transparent mass, with perhaps a faint grayish tint. In the lungs it usually assumes a more decided grayish tint, from the admixture of black pigment. This is less distinctly observed in other organs.

Can you judge, from the appearances noticed after death, whether the development of the disease has been rapid or otherwise? When the disease is in its early stage, you cannot usually form any decided opinion. Yet, in certain extreme cases, the difference in an acute and in a chronic case is very striking. When a large and sudden deposit has taken place, and death has rapidly supervened, you will find the lungs studded throughout with small whitish, opake masses, moist and pasty, while the whole lung is in a state of hyperemia, and moist with serous infiltration; while in chronic cases, you will find the lung dry, free from congestion, and tubercular masses dark and shrivelled, and surrounded with much black pigment. But in a great majority of cases this broad distinction is not so apparent. When the disease has advanced to its last stage, and cavities have formed in the lungs, if these cavities are large or numerous, and without an organized lining membrane, with evidences of recent inflammatory action, then you may believe that the case has advanced rapidly, at least in the later periods of its progress. In very chronic cases, you will find these cavities lined by a thick, whitish, fibrous membrane; partially contracted, secreting but

imperfectly developed cells, and that this imperfect development is owing to a deficiency of the phosphate of lime in their composition. A distinguished physician of New Orleans regards this as an indication of treatment.

little purulent matter, surrounded by portions of lung of a grayish or reddish hue, and condensed by chronic inflammation, presenting, perhaps, chalky concretions, with a copious deposit of black pigment—the lung being closely attached to the ribs by thick, firm, and well-organized pleuritic adhesions. When these conditions exist, you may infer that the case has been chronic in its progress, and has worn out the vital powers only after a struggle of years.

It is not unusual, however, even in these extremely chronic cases, for a copious deposit of miliary tubercles to occur, a short time before death, and to be found on post-mortem examination, especially in the lower lobes of the lungs.

Although I am speaking to you of tubercles in the lungs, yet I must not forget to remind you that the scrofulous diathesis, or constitution, eminently predisposes to the same deposit in other organs. Indeed, you must never think of the disease as existing in any particular organ, without remembering the tendency to general tuberculization, which so frequently exists. It is not my intention to enter upon this extended subject. I will only remark, that tubercle is the same in its nature and in its progress wherever it is deposited, tending more or less rapidly to the destruction of the parts affected, and attended by phenomena which, allowing for the difference in the functions of different organs, are so similar, that they serve mutually to illustrate each other. I will simply state, in conclusion, that the tuberculous deposit is found within the cranium, especially in children. Deposited, as it most frequently is, in the membranes, it produces the disease long known as acute hydrocephalus. It is still more frequently found in the lymphatic glands, in those of the neck, of the bronchi, of the mesentery. It is found also in the liver, the spleen, the kidney, the bladder, the uterus, the heart, and in the structure of the bones. Indeed, perhaps, there is no organ in the body entirely exempt from its ravages.

Louis has established a most important law, derived from the careful study of the pathological anatomy of tuberculization. When the tuberculous deposit exists in any organ, it always exists also in the lungs. The exceptions to this law are very few, especially in adult life. Among children, the exceptions

are more frequent. You must not suppose from this statement, that the deposit is, necessarily, more developed in the lungs than elsewhere. This is far from being always true; yet it is generally true, and it leads to a very important practical conclusion. Whenever the existence of tubercles is suspected in any organ, the lungs should always be carefully examined. The superiority of the means of physical diagnosis, as applied to these organs, will often enable you to establish the true character of the disease.

LECTURE XV.

TUBERCLES IN THE LUNGS.

Causes of tuberculous phthisis.—Influence of hereditary predisposition, of age, sex, professions.—Influence of climate; malaria.—Inflammatory diseases of the lungs.—Intemperance.

I SHALL occupy your attention to-day by a consideration of the causes of tuberculous phthisis. The subject is one of the greatest interest, and much labor has been bestowed in its investigation. Inquiries of this kind are, necessarily, difficult and perplexing, and sometimes lead to contradictory results. Although the observations which have thus far been made will not enable you to form such certain and definite conclusions as if the reputed causes of the tuberculous deposit were all established facts, capable of being stated as distinct propositions; yet it is well that you should know what has been accomplished, that you may be guided in the proper management of this most important disease.

Among the influences which seem to promote the deposition of tubercles in the lungs, the *hereditary predisposition* occupies a most important place. Yet this hereditary tendency explains nothing as to the real causes of the disease. It must be regarded as the simple statement of the fact, that those whose parents and blood-relatives have been the victims of tuberculous disease are

more liable to be attacked than those whose relatives have exhibited no such tendency. This hereditary influence has been questioned by some recent observers. Dr. Walshe, of London, states in his Report that 21 per cent. among males, and 37 per cent. among females, come of phthisical parents; yet he is disposed to doubt the hereditary influence of the disease. He selected 284 patients free from any suspicion of phthisis, and found that nearly 17 per cent. sprang from a phthisical father or mother. Again, he states that, among consumptive patients, the proportion who had parents free from phthisis was 57 per cent.; while, on the other hand, among non-consumptive patients, the proportion of non-consumptive parents was 47 per cent.

There are several sources of error, I think, in this statement of Dr. Walshe. In the first place, I believe that the proportion of consumptive patients with consumptive parents, will always be far below the real truth in all Hospital Reports. There are inherent difficulties in ascertaining the truth, and which can never be overcome. The patients who enter a Hospital are unknown to the physician, and their families are equally unknown. He must depend, in most instances at least, upon their statements alone. Now, Hospital patients, as you all know, are proverbially ignorant of their own antecedent diseases, and are often sadly at fault in remembering even the time and circumstances of their present attack. They seldom know how their parents were affected, or of what disease they died. They may often suppose that they died of consumption, a term which, in popular language, embraces different diseases; or, what is more probable, they will be ignorant of, or disposed to conceal the fact, when it has actually existed. Besides, I do not think that it is by any means necessary that parents alone should be taken into the account in estimating the hereditary influence of phthisis. The disease may not have appeared in either parent, and yet have shown itself, most conspicuously, among the blood relatives, the uncles or aunts, of the patient. In this instance, the family predisposition is still more difficult to be detected in Hospital patients.

Again, Dr. Walshe selects 284 persons free from phthisis, and

finds 47 per cent. without phthisical parents. Yet many of these, had they lived long enough, might have eventually died of phthisis: as the case now stands, the proportion is 10 per cent. less than among consumptive patients.

In the recent Report of the London Hospital for Consumption, you will find the same subject investigated. The proportion of consumptive patients descended from consumptive parents is 18 per cent. for the males, 36 per cent. for the females. In this report, as well as in that of Dr. Walshe, the greater relative proportion among females is very striking; not, probably, because the hereditary influence among females is any greater than among males, but simply because females, among the lower classes, being more constantly at home, are better acquainted with the family history than males.

Briquet, physician to one of the Paris Hospitals, has also published his observations on the hereditary influence of phthisis. Of 98 cases, 30 were born of phthisical parents, nearly 30 per cent., a proportion not materially different from that given in the London Reports. Yet in this statement, although the proportion is somewhat greater, perhaps owing to the greater natural intelligence of the Paris Hospital population, it is, I believe, far from the real truth. The experience of Hospital practice when compared with the observation of private practice, will always give a low estimate of the hereditary influence in phthisis. I am unable to offer any statistical results of private observation, but I am very well satisfied that a very large proportion of phthisical patients (at least 75 per cent.) belong to consumptive families. Even in this class of society, in which superior intelligence on the part of the patient, a more extended intercourse with different members of the family, as well as the personal experience of the physician in the family history, enables the private practitioner to ascertain the facts with much greater accuracy; still, unless you are careful, you will often be led into error. There is a tendency to deny, or at least to pass slightly over, this fact of family predisposition to disease. I have often ascertained that the disease I am now considering, did exist in families in which its existence had been positively denied, in the first instance. You will find, also, that while the existence of

the disease in the family is reluctantly admitted, yet, that some excuse is found for it in bad habits, or in uncommon exposure, or in some other special cause. My own opinion is, that the hereditary influence in phthisis is a well-established fact, and that the degree of this influence has an important practical bearing. That is to say, when both parents have died of phthisis, or in families in which phthisis is prevalent, the chances of an attack are not only greater, but the prognosis, when patients are attacked, is more unfavorable.

It is also probable, but the fact is not yet satisfactorily established, that children born of parents actually affected with tuberculous disease of the lungs are more likely to be attacked, and to be attacked earlier in life, than where this condition has not actually been developed at the time of birth. The Report of the London Hospital for Consumption tends, also, to establish the opinion, that phthisical fathers more frequently transmit the disease to their sons (60 times in 100 cases of transmission); while mothers more frequently transmit the disease to their daughters (57 in 100 cases of transmission).

The observations of Louis have led him to believe that phthisis is more common among females than among males, in the proportion of 70 to 57. In support of this opinion, he adduces the opinion of Benoiston de Châteauneuf, who reports that among about 1500 phthisical patients in the Hospitals of Paris, the proportion of females was $\frac{1}{3}\frac{1}{1}$ to that of $\frac{1}{3}\frac{1}{5}$ for males, of the whole number of deaths. This opinion is also strengthened by the report of Papavoine, from the Hospital for Children between the ages of one and fifteen years; in which, in a large number of post-mortem examinations, tubercles were found in three-fifths of the girls, and only in two-thirds of the boys.

But other Hospital reports contradict this opinion. Thus, in the London Hospital for Consumption, the proportion stands sixty-one males to thirty-eight females, of those attacked; while in Briquet's Report from one of the Paris Hospitals, the proportion of deaths from phthisis was just equal for the two sexes. In Lombard's cases, obtained from the registers of the city of Geneva, the proportion of men to women was as 115 to 106.

You may infer, I think, from what I have stated, that the in-

fluence of sex in the development of phthisis is not worth any serious consideration.

The influence of *age* on the comparative frequency of phthisis is very remarkable. A very large proportion of those affected, die between the ages of twenty and thirty years. Dr. Walshe includes forty-one per cent. of the whole number between these periods. Briquet states that three-fifths of those who suffer from the disease are attacked between the age of twenty and thirty-five years. Children are by no means exempt from its ravages. While all admit the rarity of tuberculous deposits in the lungs of the fœtus, still they are sometimes noticed. I have seen the lungs of a new-born infant completely studded with tubercles. Although the tendency to the disease rapidly diminishes after the age of thirty or thirty-five years, yet it is sometimes met with even in advanced life.

Sir James Clark places the mortality between the ages of fifty and sixty years at 108, as compared with 285 representing the mortality between the ages of twenty and thirty years. A table exhibiting the relative age when attacked with the disease, would be still more interesting, for by this you would learn that very many of those who died at an advanced age had been attacked by the disease at a comparatively early period in life. My own impression is, that when individuals who have passed the middle period of life are attacked with phthisis, the disease progresses less rapidly than at an earlier period of life, and that many thus die, after having suffered from the disease for a very considerable portion of their lives.*

In estimating the causes of phthisis, you would naturally be led to ask the question, How far do the *occupations of life* influence the production of the disease? We possess some valuable statistical information on this subject, especially from Lombard

* The following statement will illustrate the relative influence of age as to the frequency of phthisis:

Paris.—1st period of frequency, from 20 to 30 years; 2d, from 30 to 40 years; 3d, from 10 to 20 years; 4th, from 40 to 50 years; 5th, from 50 to 60 years; 6th, from 0 to 10 years; 7th, from 60 to 70 years.

Consumption Hospital, London.—1st period of frequency from 25 to 35 years; 2d, from 15 to 25 years; 3d, from 35 to 45 years; 4th, from 45 to 55 years; 5th, from 5 to 15 years; 6th, from 55 to 65 years; 7th, from 0 to 5 years.

of Geneva, derived from the Registers of that city. The final results of his researches are thus stated: "The circumstances which increase the tendency to phthisis are poverty, sedentary habits, violent exercise of the chest, an habitually bent position of the body, impure air in workshops, the inhalation of certain mineral and vegetable vapors, or air loaded with a coarse or impalpable dust, or with light, thready, elastic substances.

"The circumstances which seem to exert a favorable, a preservative influence, are easy circumstances, an active life in the open air, regular general exercise, the inhalation of watery vapor, and finally, animal and vegetable emanations."

This general summary of the results of a very extended and careful examination of the subject, is worth considering. We have but one similar series of observations with which to compare it, that of Benoiston de Châteauneuf, made at Paris, in the different Hospitals of that city, at about the same time that Lombard was engaged in his labors at Geneva, and without any knowledge of the results at which he might arrive.

The unfavorable influence of poverty and its accompanying evils, exposure, anxiety, bad nourishment, seems to be admitted by all observers, and yet they have been unable to appreciate, with any degree of accuracy, the effects of these agents upon the disease in question—inasmuch as they seldom or never exist alone. Lombard estimates the proportion of deaths from phthisis in those professions practised by the higher classes of society as only one-half as great as among the poorer classes. In Geneva, the proportion of deaths from phthisis among those living upon their incomes, is only fifty in one thousand deaths, while the average number of deaths from phthisis in all classes is one hundred and fourteen in one thousand deaths.

Sedentary habits, especially the habit of sitting constantly with the body inclined forward, seems to exert a decided influence in the development of phthisis; the proportion of cases among shoemakers and tailors being very large. The effect of this position upon both digestion and upon respiration is no doubt very considerable.

The unfavorable influence of violent exercise, especially of the chest, is a fact by no means established. The same opinion

may be formed of the influence of an atmosphere charged with aqueous vapor. Lombard and Benoiston de Châteauneuf have arrived at quite opposite conclusions on these points.

The influence of the inhalation of various substances by the lungs is a point of great interest. These influences are of different kinds. Some of them, as the inhalation of gases and vapors, seem to exert their influence by absorption. Others, again, exert their influence directly upon the lungs by acting mechanically upon their tissues. It does not appear that the first class of inhalations, however much they may influence the general health, exert any special influence on the pulmonary tissues, except the inhalation of the vapor of varnish and of the drying oils. Those exposed to the influence of the vapor from mercury, lead, and other mineral agents, are not particularly liable to phthisis, unless, perhaps, those who are exposed to the mercurial influence. This latter question is one of great importance, when you remember that the general impression exists in the profession, that mercurials are injurious to phthisical patients. The observations of Benoiston de Châteauneuf tend to confirm this opinion. Among gilders, who are exposed to mercurial vapors, the proportion of deaths from phthisis is fifty-three in one thousand; while among painters, who inhale the vapor of lead, the mortality from phthisis is only twenty-one in one thousand. The statement, however, of Lombard does not agree with this conclusion. His observations, on the contrary, tend to prove that phthisis is not a common disease among gilders.

The inhalation of dust, or of distinct particles from animal, vegetable, and mineral substances, has also been carefully studied in its influence upon the production of phthisis. While all admit their injurious effects, it is by no means clear to my mind that they produce tuberculous phthisis. They probably produce a form of chronic bronchitis, of a disorganizing and fatal character, rather than the deposit of tubercles. Neither are observers agreed as to the relative influence of these different irritants. Lombard thinks that mineral dust is the most injurious, then animal dust, and least of all, vegetable dust. While Benoiston de Châteauneuf is of the opinion that animal

dust is the most injurious. It is difficult to classify these agents, as to their influence upon the lungs, according to their source.

Those agents which experience has proved to exert the most deleterious influence are, the dust arising from the manufacture of flints and of sandstone, from the polishing of steel, among mineral agents; that arising from the dressing of feathers, hair, &c., among animal agents; and finally, that arising from the dressing of cotton, among vegetable agents.

It will also appear probable that the finer particles of dust are more injurious than those of a larger size.

Among the favorable influences, the preservative influences against the encroachments of phthisis, the good effects of easy circumstances, of an active life in the open air, will generally be admitted. But when you are told that constant exposure to watery vapor exerts also a favorable influence, you may well question the fact. That a warm and moist atmosphere may exert a favorable influence upon the lungs, will be readily admitted; but the injurious effects of moisture, combined with cold, must be evident to every one accustomed to our climate. Lombard was led to form a favorable opinion of exposure to a moist atmosphere by the fact, that the washerwomen of Geneva are but little disposed to phthisis. But the observations of Benoiston de Châteauneuf at Paris have led him to quite an opposite conclusion. It is also the opinion of M. Lombard, that a warm and *dry* atmosphere exerts an unfavorable influence. Animal and vegetable emanations are also regarded by Lombard as exerting a favorable influence upon this disease. Butchers, tanners, leather-dressers, are remarkably exempt from phthisis. But with regard to vegetable emanations, the truth of Lombard's statement may well be doubted. In certain regions, the healthful influence of vegetable emanations may be true enough. But if the various forms of malarious disease are dependent upon this cause, then we must class these emanations among the most unfavorable influences in the production of phthisis. There is, indeed, a popular opinion in this country, that a residence in a malarious region is favorable to phthisical patients. But this opinion is the very reverse of the truth. Malarious diseases, by impairing the

general health, favor the development of phthisis, and much increase its mortality.

The free exercise of the voice is also regarded by M. Lombard as exerting a favorable influence.*

It is generally supposed that a feeble constitution predisposes to the development of phthisis. There is reason to doubt the truth of this received opinion. At all events, the disease is usually less rapid in its progress when it attacks those of a delicate constitution; and a knowledge of this fact would naturally lead you to doubt the influence of this condition in the development of the disease. Briquet states, that thirty-three of his patients were of vigorous constitution, twenty-one were delicate, the remainder, one hundred and twenty-eight in number, possessed medium constitutions.†

We are indebted to the valuable reports of the British army surgeons and to those of the late Dr. Forry of the United States

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* The following table from Lombard's Essay, presents a general summary of favorable and unfavorable influences in the production of phthisis: 114 in 1000 deaths is taken as the mean mortality in phthisis for all classes or professions.

Influences favoring the development of phthisis:

1. Mineral and vegetable emanations	0·176	(176 in 1000 deaths).
2. Dust of different kinds inhaled	0·145	
3. A sedentary life	0·140	
4. A life passed in workshops	0·138	
5. A warm and dry atmosphere	0·127	
6. A stooping position of the body	0·122	
7. Shocks to the chest from violent movements of the arms (?)	0·116	

Influences unfavorable to the development of phthisis:

1. An active life	0·089	(89 in 1000 deaths).
2. Exercise of the voice	0·075	
3. Life in the open air	0·073	
4. Animal emanations	0·060	
5. Watery vapor (?)	0·053	

† Louis is of the opinion that the lymphatic temperament predisposes to phthisis. Papavoine found in 298 children, between the ages of 2 and 15 years, that 74 were of dark complexion, 115 were blonds, and 129 were intermediate (chestnut hair). Briquet in his report states that in 106 patients, adults, male and female, 29 were dark, 14 blond, and 55 medium (chestnut). But an attempt to distinguish the different temperaments, especially by the color of the hair and of the complexion, could hardly lead to any certain conclusions.

army, for very important information in relation to the prevalence of phthisis in different climates. These reports have corrected some striking mistakes on the subject, errors of much practical importance. It has been thought that the disease prevailed chiefly in what are called temperate latitudes, in the central portions of Europe, and in the northern and in the middle portions of the United States. It has been supposed that the surest protection against the ravages of the disease was a removal to, and a permanent residence in, a tropical climate. But while the great mortality of the disease in temperate latitudes is still admitted, the most important practical fact has been ascertained, that a permanent residence in a tropical climate, so far from diminishing the chances of death from phthisis, tends rather to increase the chances of this result. This remark does not, indeed, apply to all tropical countries which have been made the subject of study. Some regions, indeed, seem to exert a favorable influence. There is a great difference, for instance, in this respect, between the East and the West Indies, the climate being much more favorable in the former region than in the latter. We do not know the relative mortality from phthisis in temperate and in tropical regions among the native population of those regions; neither is this a point, at all comparable in importance, to the fact that the natives of temperate regions who reside permanently in tropical climates are less protected from the ravages of the disease than those who remain at home. We who live and practise our profession in temperate regions are not unfrequently asked the question by consumptive invalids, whether they should leave this region of country and settle in a tropical region? certainly not, if the question is to be answered in a general way: perhaps so, if certain localities are selected. I cannot help thinking that a more particular statement of the facts of the case will be of service to you.

The British Army Reports embrace a statement of the diseases of soldiers stationed in different quarters of the globe, where they usually remain for several years. The standard of comparison is the Dragoon Guards and Dragoons stationed in Great Britain, who have been selected as corresponding more nearly with the troops in the Colonies, in all other respects ex-

cept in climatic influences, than any other body of troops in Great Britain. The number of these troops attacked with consumption, annually, in Great Britain, is estimated at six and one-half of every thousand men. The mortality from the disease may be estimated pretty nearly by the same figures, while the annual mortality from all diseases of the lungs, reaches only to about eight in one thousand men. Now in the West Indies, in the Leeward and Windward Islands, the proportion of cases attacked with phthisis, annually, is twelve in one thousand men. In Jamaica, the proportion of cases of phthisis treated annually, reaches as high as thirteen in every thousand men. In the Bermudas, the proportion of those attacked is nine in one thousand men.

If, now, we turn to the Reports from the Mediterranean Stations, we shall find an improved state of the question, but yet not so favorable a report as might have been anticipated. In Malta, nearly seven in one thousand men are attacked annually. In the Ionian Islands the proportion is somewhat less, five to six of one thousand men being annually attacked; while in Gibraltar the mortality reaches only to about five in one thousand men. But this small mortality is explained by the fact, that many of the Gibraltar invalids are sent home. Formerly, before this was done, the mortality reached as high as twelve in one thousand men.

If we now turn to the East Indies, we shall find a still greater improvement in the results. Indeed, there can be no doubt that the question of permanent removal, which would be answered in the negative for the West Indies and the Mediterranean, might be answered in the affirmative for the East Indies.

In Ceylon the proportion of those attacked with phthisis, annually, is only about five in one thousand men, the proportion of deaths being about three in one thousand men. In the British Possessions, extending along the eastern shore of the Bay of Bengal, the report is still more favorable; but four or five cases of phthisis have occurred there in as many years.

In Mauritius, on the other hand, the proportion of those attacked is much increased, being, annually, nearly eight to every thousand men.

If, now, we turn to more northern regions, to the cold regions of the globe, we shall infer from the imperfect statistical information we possess, that phthisis is comparatively a rare disease in these regions. In Sweden, the ratio of deaths from phthisis, in one thousand deaths, is only sixty-three, while in London it is two hundred and thirty-six.

In Canada, according to the Army Reports, only $6\frac{5}{10}$ in 1000 men are attacked annually, a remarkable result when compared with that obtained in the West Indies: a result which, if I am correctly informed, has induced the British Government to send their consumptive soldiers from the West Indies to Canada, as affording them the best chances of life; the proportion of those attacked annually, even in England, being at least one greater in every thousand men, and being at least double in amount in the West Indies.

The very able Report of the late Dr. Forry, founded on the statistics of the U. S. Army, at the different military posts scattered over our widely extended country, confirms the opinion formed by examining the British Reports. Contrary to the general belief, phthisis is found to be more fatal among the troops stationed along the southern Atlantic coast, from Delaware Bay to Savannah, and at the southwestern posts, than at the northern Atlantic posts, and at those situated on the great lakes. While the least ratio is found at those posts in the northern division of the United States, remote from the ocean and the lakes. Thus, so far as our own country is concerned, the regions least predisposing to phthisis, are the inland States in the northern division, removed equally from the influence of the ocean and of the large lakes.*

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* The following table from Dr. Forry's work will serve to illustrate the above remarks.

Phthisis Pulmonalis.—Ratio of cases per 1000 of mean strength.

Northern Division.	Atlantic posts	9	of 1000	men.
	Posts on the lakes	9	"	"
	Posts remote from the ocean and lakes	5	"	"
Southern Division.	Coast from Delaware to Savannah	13	of 1000	men.
	Southwestern stations	11	"	"
	Posts on Lower Mississippi	9	"	"
	East Florida	9	"	"

In attempting to estimate the influence of climate on the development of phthisis, you must be struck at once with the fact that parallels of latitude are very unsafe guides in judging of this question. But there is more than this. Places situated in nearly the same parallels of latitude, give very different results. Neither will the variations of the temperature at different seasons, or even for shorter periods, months, for instance, explain this difference; neither does the annual quantity of rain which falls.*

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* *Difference in the mean temperature of the coldest and warmest months, and in the mean difference of the successive months.—Quantity of rain.*

Southern Atlantic Posts.....Mean diff. summer and winter, 30 to 35°
" " " .....Mean diff. of successive months, 5 to 6°
Quantity of rain, 40 inches annually.
Southwestern Posts.....Mean diff. summer and winter, 27 to 42°
" " .....Mean diff. of successive months, 4 to 7°
Quantity of rain, 30 to 51 in. annually.
Inland Posts, remote from the Lakes...Mean diff. summer and winter, 45 to 54°
" " " " Mean diff. of successive months, 7 to 9°
Quantity of rain, 24 to 30 in. annually.
Northern Atlantic Posts.....Mean diff. summer and winter, 41 to 47°
" " " .....Mean diff. of successive months, 6 to 8°
Quantity of rain, 28 to 47 in. annually.
Lake Posts.....Mean diff. summer and winter, 49 to 54°
" " .....Mean diff. of successive months, 7 to 9°
Quantity of rain, 28 to 38 in. annually.
Florida.....Mean diff. summer and winter, 14 to 28°
" .....Mean diff. of successive months, 2 to 4°

In the West Indies:

Windward and Leeward Islands.....The mean annual temperature is 80°
" " " ..... extreme range.....13°
Quantity of rain, 60 to 70 inches.
Jamaica (Kingston).....The mean annual temperature is 80°
" " ..... extreme range,.....11°
Quantity of rain, 50 inches.
Bahamas.....The mean annual temperature is 78°
" ..... extreme range,.....10°

In the East Indies:

Ceylon.....The mean annual temperature is 80°
" ..... extreme range at Columbo, 14°
" ..... " " Trincomalee, 15°
" ..... " " Kandy, 24°
Quantity of rain, 50 to 80 inches.

The influence of *malaria* seems to be more clearly indicated. This poison, especially when acting with a medium intensity, producing intermittent and remittent fevers which do not rapidly destroy life, but which gradually undermine the constitution, seems to lay the foundation of phthisis in those who are predisposed to the tuberculous deposit. If we knew the comparative frequency of phthisis among the native population of tropical regions, we might understand the influence of climatic causes much better; but on this point we possess very little information. In the West Indies, phthisis is much less prevalent among the officers than among the men, while fever is equally prevalent, but the men are much more subject to bowel complaints.* In Ceylon, where phthisis does not prevail, fever is very prevalent. At Mauritius, diarrhœa and dysentery are also very prevalent.

When we turn to the statistical reports of our own army surgeons, we find that malarious diseases are, as a general rule, most prevalent in those regions which yield the largest proportion of cases of phthisis, yet the ratio is not uniform. Thus, if we omit the posts in New England, where this class of diseases hardly exists, we shall find that at the inland posts, where phthisis is the most rare, malarious diseases are also least frequent, while at the southwestern posts, and at those from Delaware Bay to Savannah, where phthisis is most prevalent, malarious diseases are also most prevalent. But in Florida this ratio is not observed. In this region phthisis occupies a medium place, while the ratio of malarious diseases is high; the reverse of this is found to exist at the posts on the great lakes.†

* The prevalence of stomach and bowel affections in the West Indies is indicated by the following statistics:

Windward and Leeward Islands, number attacked annually,				421 in 1000 men.
Jamaica	"	"	"	238 in 1000 "
Bermuda	"	"	"	415 in 1000 "
In Great Britain	"	"	"	94 in 1000 "

† *Ratio of intermittents, remittents, diarrhœa and dysentery in every* 1000 *men treated annually.*

	Intermit.	Remit.	Diar. & Dys.
Posts on the coast of New England	36	26	170
" " Northern Lakes	193	33	253
" remote from the ocean and Lakes	151	24	305
" on the coast from Delaware Bay to Savannah	370	181	455
" Southwestern	747	180	597
" Lower Mississippi	385	196	456
" East Florida	520	102	495

The favorable influence of a *sea life* on the development of phthisis has long been recognized, and this opinion is confirmed by the observations of the British naval surgeons. A comparison of the Reports for the North American and West India Stations, gives only five attacked in every one thousand men, and a mortality of only two in one thousand men.

It may be remarked, however, that while sailors and soldiers are equally examined as to their actual health at the time of enlistment, and none are enlisted except those supposed to be in good health, yet sailors are enlisted for a much shorter period of time than soldiers, and the chances of being sent home as invalids are much greater. Both these circumstances are capable of diminishing very much the proportion of deaths from phthisis among sailors, as exhibited by the statistical reports of the naval medical officers on active duty.

It has long been the popular belief among medical men, that *the acute inflammations of the chest* exert a decided influence in the production of tuberculous phthisis. If you listen to the reports of patients, they will often trace the disease back to some neglected cold, or to some acute attack neglected or badly treated. I think that there is but little real foundation for this opinion. In the first place, if you look at the question in a pathological point of view, you will find but little to confirm the general opinion. Thus you will remember the fact, that tubercles are frequently found developed in lungs quite free from inflammation, and that the deposit in the more external portions of the body, and which in its primary stage can be more exactly watched—as in the lymphatic glands of the neck, for instance, exhibits no early indications of inflammatory action. If now you examine the seat of these different diseases as tending to illustrate their influence on each other, as cause and effect, you will find that tubercles are seated in the upper lobes of the lungs, at least during the early progress of the case. Bronchitis attacks the lower lobes of the lungs. Pneumonia also attacks the lower lobes, at the period of life when tubercles are most apt to occur, while later in life, when the disposition to the deposit of tubercles is comparatively rare, pneumonia of the upper lobes is comparatively common. Pleurisy is a disease more extensive

in its seat, spreading over the whole surface of the lungs, and I am inclined to think that the dependence of tubercles on pleurisy is better established than in the case of pneumonia and of bronchitis. I think you will not unfrequently meet with tuberculous disease of the lungs which has followed an attack of pleurisy. But it may be questioned whether in such cases the deposit may not have existed in a latent state before the attack of pleurisy occurred.

Again, if bronchitis and other inflammatory diseases of the chest were a prominent cause of the tuberculous deposit, you ought, I think, to find them observing a certain ratio with phthisis in the statistical reports of pulmonary diseases. In those regions where phthisis is most prevalent, these inflammatory diseases ought also to be most prevalent. But it is not so. They are different entirely from the malarious diseases in their ratio to phthisis. Thus, in Dr. Forry's Reports, bronchitis is most prevalent at those posts remote from the ocean and the lakes, precisely where the ratio of phthisis is the least. In relation to pneumonia and pleuritis, the difference is far less striking; but even in this case, there is no definite ratio in the relation of these diseases to phthisis; that is to say, phthisis is by no means most prevalent at those stations where pneumonia and pleurisy are most frequent. I cannot help thinking, however, that had the cases of pleurisy and of pneumonia been carefully discriminated and judged separately, which is not attempted to be done, a different result might have been obtained. I think it might have appeared that, while the ratio of pneumonia, like bronchitis, exhibited no relation to phthisis, that the ratio of pleurisy might have exhibited such a relation.*

The British reports confirm the conclusions of Dr. Forry; and

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* Ratio of bronchitis, pneumonia, and pleurisy, annually treated in 1000 men:

	Bronchitis.	Pneumonia and Pleurisy.
Posts on Coast of New England	233	41
" on Great Lakes	300	49
" Remote from Ocean and Great Lakes	552	45
" From Delaware Bay to Savannah	271	57
" Southwestern	290	92
" Lower Mississippi	218	47
" Florida	143	89

it is a striking fact, that, while phthisis is comparatively rare among sailors, bronchitis is twice as frequent as in the army.

It is an important question to answer, How does the intemperate use of ardent spirits influence the production of phthisis? This is a delicate question; but I must state what I know about it. Two medical gentlemen attached to the public Dead-house in this city, in which bodies are deposited which are found in the streets, or without friends—discovered in about seventy post-mortem examinations of those who had died of the most confirmed and aggravated intemperance, not a single case of tuberculous lungs. A most surprising result, when you remember that this unfortunate class have, probably, long suffered from poverty, bad nourishment, and exposure to the weather; influences which are regarded as predisposing to the tuberculous deposit. It is a singular fact, also, that while phthisis is much more common in the West than in the East Indies, hepatic affections are comparatively rare.* A large proportion of confirmed drunkards suffer also from hepatic disease. Is a tendency to hepatic disease antagonistic to the development of tubercles?

The exercise of the organs of the voice appears, as I have already stated, to exert no unfavorable influence on the development of phthisis; but, on the other hand, the influence is, probably, beneficial. But when the disease is once established, the most injurious effects may result from this cause.

Dyspeptic affections have been regarded as exerting a most decided influence in the development of phthisis. Dr. Todd, of London, has described a form of dyspepsia which he calls *strumous*, and as characterized particularly by congestion of the

* In the West Indies, Windward and Leeward Islands, the cases of hepatic disease treated annually are 22 in 1000 men. Deaths, 2 in 1000 men.

In Jamaica, the mortality from hepatic disease is only 1 in 1000 men; and in Bermuda, only $^{5}/_{10}$ in every 1000 men.

In the Mediterranean, the mortality from hepatic disease is also very small. In the Ionian Islands, it is only $^{6}/_{10}$; at Gibraltar, $^{4}/_{10}$; and at Malta, $^{11}/_{10}$ in every 1000 men.

In the Eastern tropical regions, the proportion of hepatic disease is much greater.

At Mauritius, 82 in 1000 men are attacked annually. Deaths, 4 in 1000 men.

At Ceylon, 55 in 1000 men are attacked annually. Deaths, 5 in 1000 men.

portal circulation. That imperfect assimilation of the food is the direct cause of the tuberculous deposit, is a favorite theory to which I have already alluded. That certain occupations characterized by confinement to bad air, sedentary habits, and especially the habit of stooping forward, which exert an unfavorable influence on phthisis, also predispose to dyspepsia, and perhaps, in the first instance, produce a derangement of the digestive functions, are facts sufficiently well established. Still, nothing is more common in phthisis than to meet with individuals who have not suffered, at least at the time immediately preceding the first phthisical symptoms, with any marked form of dyspepsia. I am inclined to think that the later English writers on this subject have overrated the influence of this cause.

In the somewhat extended examination of the causes of phthisis, which I have now concluded, I have been careful to avoid any speculative points, preferring to present to you certain statements which have been made on a sufficiently extended scale, and with sufficient accuracy, to be regarded at least as an approximation to the truth. You have been told that the disease is most frequent at a certain period of life; that it attacks the two sexes in about equal proportions, males perhaps rather more frequently than females; that it is often hereditary; that no peculiar constitution of body or temperament is more liable to an attack; that certain professions or occupations are more liable to induce it than other occupations; that a continued residence in tropical climates does not exert a favorable influence on the disease, as it exists in the natives of temperate regions; that a sea life seems to exert a beneficial influence, especially in warm latitudes; and, finally, that neither the exercise of the vocal powers, nor the existence of acute inflammations of the chest, nor habits of gross intemperance, seem to exert an influence favorable to the development of tuberculous phthisis.*

* By the Report of the Inspector of the City of New York, and by similar Reports which I have obtained from some of the other cities of the Union, I find that the proportion of deaths from Consumption, to the whole population of those cities, is the following:

Boston	1 in 236	Baltimore	1 in 290
New York	1 in 267	Charleston	1 in 426

By comparing the mortality from Consumption with the general mortality, I obtain the following results:

Boston	(period of 5 years)	1 in 6·6
New York	(" " 4 years)	1 in 7·2
Baltimore	(" " 10 years)	1 in 5·4
Charleston	(" " 5 years)	1 in 6·9

In this calculation, casualties, as death by drowning, suicide, &c., are excluded, also premature and still-born children, except in the case of Boston and Charleston. If the Reports from these cities had permitted me to exclude the premature and still-born, it would have increased considerably the relative ratio of deaths in these cities.

In New York, during two years, the greatest number of deaths from Consumption occurred during the month of January, viz., 423. The least number of deaths occurred in June, viz., 254.

The most fatal months, and in the order of their fatality, were:

1848. January, February, March, April, October, December.
1850. January, December, November, October, March, August.

The least fatal months, and in the order of their least fatality, were:

1848. September, June, November, July, May, August.
1850. June, July, February, May, September, April.

1848.	The mortality from Consumption among males was...	946
1850.	" " " " "	982
		1928
1848.	The mortality from Consumption among females was,	923
1850.	" " " " "	949
		1872

The proportion of deaths from Consumption, according to age, is as follows:

	1 yr.	1 to 2 yrs.	2 to 5 yrs.	5 to 10 yrs.	10 to 20 yrs.	20 to 30 yrs.
1848	62	41	55	31	133	516
1850	76	63	55	28	144	559
	138	104	110	59	277	1075

	30 to 40 yrs.	40 to 50 yrs.	50 to 60 yrs.	60 to 70 yrs.	70 to 80 yrs.
1848	459	282	164	69	31
1850	435	274	143	88	35
	894	556	307	157	66

According to the Report of the Registrar-General for 1842, the proportion of deaths from Consumption in London, to the whole population of that city, is one in 262. The proportion of deaths from Consumption in London, to the whole number of deaths in that city, is 7·1.

In the Registrar's Report for 1840, it is stated that in England 4 in 1000 persons die annually of Consumption, and that from one-fifth to one-sixth of the whole number of deaths is from this disease.

By the census of 1845, the proportion of males to females in the city of New York is as 180,472 to 190,751.

LECTURE XVI.

TUBERCLES IN THE LUNGS.

Simple phthisis.—History of the disease.—Critical examination of the principal symptoms.—Rational symptoms.—Cough, expectoration, hæmoptysis, dispnœa, pain.—Constitutional symptoms.—Emaciation and loss of strength, pulse, hectic.—Digestive and menstrual functions.

You are called to visit a patient who has experienced, for a considerable time, for two or three months, a dry, hacking cough, attended with some emaciation and loss of strength, and with a tendency to acceleration of the pulse, especially towards evening. There exists, probably, also an hereditary tendency to phthisis, and these symptoms have created some alarm in the family, who have, very likely, watched the development of the disease in others of its members. The occurrence of these symptoms alone, even when no physical signs of disease can be discovered, ought, justly, to excite your fears, and prevent you from thinking lightly of the case, as too often happens. Frequently, indeed, other symptoms are present. A certain degree of dyspnœa exists, with flying pains about the chest, and, perhaps, an attack of hæmoptysis has occurred. Under these circumstances, the case becomes more than suspicious; and if you find under one of the clavicles a degree of dulness on percussion, however slight, accompanied by a feeble murmur, or by a prolonged expiration, or if the murmur of inspiration is harsh or jerking, or attended by a slight, dry crackle, then the diagnosis of the case, even at this early period, may be considered as established. Tubercles exist in the lung.

In the progress of the case, the symptoms assume a more marked character. The emaciation and loss of strength increase, the dyspnœa and pain in the chest become more decided, the pulse is more accelerated, the cough becomes more frequent, and begins to be attended by a mucous expectoration, at times, perhaps, mixed with blood. The results of a physical examina-

tion are also more decided. The dulness on percussion under the clavicle has become more marked, the respiratory murmur in the upper portion of the lung is more altered; perhaps it has become even bronchial, and you hear with it a mucous rattle. The vocal resonance is also increased. This increased development of the symptoms, and of the physical signs, does not, necessarily, indicate an increase in the amount of the tuberculous deposit. The tubercles originally deposited have grown larger, they obstruct the lung more—a certain amount of pulmonary congestion, also, probably exists—inflammatory congestion, with increased mucous secretion into the neighboring bronchi, marking a tendency to the softening of the tuberculous deposit. Or, on the other hand, a new deposit of tubercles may have occurred, and then you will perceive their effects, not only in the increased development of the physical signs in the lung primarily affected, but, probably, also in the opposite lung, in which a corresponding dulness on percussion has begun to exist, and in which the respiratory murmur has also experienced corresponding alterations.

As the tuberculous deposit continues to soften, the constitutional symptoms still progress—the emaciation and loss of strength, the frequency of the pulse. Hectic symptoms supervene—a disposition to irregular chills, to fever in the evening, and to night-sweats. A certain degree of hoarseness is of frequent occurrence. The digestive functions become impaired, and in females, the menstrual function ceases, or nearly so. The rational symptoms also become more decided—the dyspnœa, the tendency to pain in the chest; while the cough is more marked and is attended with a more copious expectoration—no longer simple mucus but opake mucus; and fleecy masses, or broken and jagged particles are seen floating in a quantity of white, thin, and transparent fluid.

An abscess at length forms in the lung first affected, which, if it does not communicate with a considerable bronchus, gives no indication of its existence. But if this communication is established, the dulness on percussion under the clavicle may diminish, to give place to a peculiar resonance, different from the healthy resonance, and sometimes resembling the cracked-pot sound.

The respiratory sound becomes cavernous, the mucous rattle becomes a gurgling, the resonance of the voice is much increased. Listening lower down in the chest, you will find that the deposit has extended to that portion, perhaps nearly or quite to the base of the lung. You will notice that the respiratory murmur is altered in the same region, while, perhaps, the mucous rattle is heard there. In the opposite lung also, the evidence of progress in the disease, and of new deposits is very apparent, and is marked by the same changes in the physical signs as in the lung first affected. The rational symptoms continue, the expectoration becoming more abundant, and assuming a more purulent character. The emaciation, the debility, the tendency to night-sweats, and to febrile excitement, the feebleness of the digestive organs, with, perhaps, a tendency to diarrhœa, are in continual progress, until at length exhausted nature sinks, and the patient, gradually suffocated, expires. Such is the history of a case of simple phthisis.

Let me now, having examined this simple case, endeavor to estimate the value of the leading symptoms somewhat in detail. I know of no better method than this to arrive at a correct understanding of disease. In the first place, then, let me discuss the rational symptoms.

I have spoken of the cough as one of the earliest symptoms of phthisis. Not a prominent and distressing cough, but a single, dry hack, often supposed by the patient to depend on irritation in the throat. This cough may be so trifling, that I have known a physician kept in ignorance of it for several weeks in the case of one of his own children, whose health he was anxiously watching. It must be confessed that the cough has not always this character. It sometimes begins in a more decided way, and is early attended with expectoration. In the advanced stage of the disease, when the secretion from the bronchi has become abundant, the cough is more prominent. Many patients cough but little, except in the morning after rising, when a long, and often violent paroxysm, sometimes occurs. Other patients experience several paroxysms during the twenty-four hours, with intervals of rest; while sometimes, especially in the advanced stage, there are no such intervals.

The expectoration should be carefully noticed in this disease. At first, trifling in amount—only a little transparent, frothy mucus, it keeps pace with the cough and gradually assumes new characters. While the tuberculous matter is softening, the mucus expectorated becomes more opake from the attending bronchial inflammation—it often presents little opake, whitish masses, fleecy, rolled up upon themselves; or jagged portions of more or less opake mucus, floating in a considerable quantity of thin, transparent fluid. This appearance of mucus, broken up or rolled up into small masses and floating in a serous fluid, is a characteristic expectoration of the softening stage of the tuberculous deposit. But when abscesses have opened into the bronchi, then the expectoration, mixed, often, with the form just described, becomes more opake and purulent, sinking in the serous fluid. Each sputum assumes and preserves a distinct form, usually more or less rounded, so that you can often count the number of expectorations by counting the number of sputa. These distinct sputa may be marked by whitish, opake lines running through them, and produced by the softened tuberculous matter which is thus undergoing expectoration. Sometimes you will find small masses, like portions of boiled rice in the expectoration: these may be occasionally tuberculous masses, but often, I think, they are only a secretion from the air-passages. It is unfortunate that the microscope does not enable you to detect the existence of tuberculous matter in the expectoration. It is, usually, too far disorganized to render this possible.

The expectoration, even in the last stage, seldom or never assumes a distinctly marked purulent character, like the pus observed in a simple abscess. Scrofulous pus seems to have a tenacious, stringy, semi-liquid character, and being mixed with more or less opake mucus in its passage through the bronchi, it assumes the characters of opake mucus, rather than those of genuine pus. This secretion will also vary much during the day. It is very common for patients to expectorate a thick, yellowish-green, opake matter in the morning, especially after a quiet night, while, during the remainder of the day, it is much less opake and yellow.

I have known several patients who have expectorated the

chalky concretions that form in the lungs. Usually this expectoration occurs in small quantity, and at rare intervals. It indicates a curative effort.

Hæmoptysis is a frequent and most important symptom in phthisis. Indeed, I may say that it throws more light on the real nature of a case in which it occurs, than all the other rational symptoms together. So rare is hæmoptysis in any other disease of the chest, that it points most unequivocally to the true nature of the case. In certain forms of heart disease, in gangrene of the lungs, in cancer, in cirrhosis of the lungs, and perhaps occasionally in women, as a form of vicarious menstruation, you may find hæmoptysis. But these cases, in addition to their peculiar attendant symptoms, are so rarely met, while hæmoptysis from tuberculous disease is so common, that its occurrence will naturally bring with it the presumption that, when hæmoptysis exists, tubercles in the lungs exist.

Hæmoptysis is sometimes a very early symptom of phthisis. It may be the very first symptom. I have known patients experience an attack without its being preceded or followed by cough, or by any other symptom. Finally, after a year, another attack of hæmoptysis occurred, still without being preceded by cough, or by other symptoms, but being speedily followed by all the well-marked evidences of phthisis. The attacks are seldom frequently repeated. Many patients only experience one attack, and sometimes only towards the close of the disease. The quantity expectorated during an attack varies very much in different cases.* It is sometimes trifling in amount, and mixed with mucus, but it is repeated at intervals for several days. At other times, and this frequently happens at the onset of the tuberculous disease, the discharge of blood is copious, even alarming in its quantity. It seldom happens, however,

* In 67 cases of phthisis with hæmoptysis, it was profuse in 29 cases; slight in 38 cases.—*Parkes.*

In 57 cases it was profuse in 25, slight in 32.—*Louis.*

Hæmoptysis:

Profuse, before softening,	35	times	in	100	cases.	
Slight " "	64	"	"	"		
Profuse, after softening,	50	"	"	"		
Slight " "	50	"	"	"		*Parkes.*

that the hemorrhage proves immediately fatal, unless it occurs at a late stage of the disease. I have, however, known a few cases in which it proved fatal at an early period.*

It is remarkable, that children before the age of puberty very seldom experience hæmoptysis. The comparative frequency of this symptom at a more advanced period of life is, perhaps, not yet fairly established. It is the opinion of some observers that women are more subject to this symptom than men, especially after the middle period of life.† The connection of hæmoptysis with menstruation is a very interesting subject of inquiry. Mistakes are often made in relation to it. The menstrual function ceases in the progress of phthisis, and hæmoptysis occurs. This is regarded as a vicarious discharge. I do not think that the relation of cause and effect is established in most cases. Now and then, you will meet phthisical females with suppression of the menses, who, at the return of their menstrual periods, in addition to other signs of internal perturbation, experience oppres-

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* Influence of the stage of the disease on hæmoptysis:

Before the softening of the tubercles, 75 in 100 had hæmoptysis.
After " " " " 26 in 100 " "
Hospital for Consumption.

Before the softening of the tubercles, 71 in 100 had hæmoptysis.
After " " " " 86 in 100 " "
Walshe.

† Influence of sex on hæmoptysis:

In females, 36 of 42 cases of phthisis (86 in 100).
In males, 21 of 38 " " (55 in 100).
Louis.

In females, 65 of 100 cases of phthisis.
In males, 62 of 100 " "
Hospital for Consumption, London.

In females, 44 in 56 cases (78½ per cent.) of phthisis.
In males, 62 in 75 " (82½ ") "
Walshe.

Influence of age on hæmoptysis:

Before puberty 2 in 100 cases.

In females (19 to 40 years), 66 in 100 cases had hæmoptysis.
" (40 to 65 "), 86 in 100 " " "
Louis.

In females (under 35 years), 67 in 100 cases had hæmoptysis.
" (over 35 years), 54 in 100 " " "
London Consumption Hospital.

In females (15 to 30 years), 73 in 100 cases had hæmoptysis.
" (30 to 50 years), 93 in 100 " " "
Walshe.

sion in the chest, sometimes followed by hemorrhage. But these cases are rare.

What are you to understand by a profuse hemorrhage from the lungs? When a patient expectorates two or three ounces of pure blood in a few hours, this may be called a free hemorrhage. When a patient expectorates less than this, an ounce, during the day, or occasionally mucus tinged with blood, it may be called a moderate, or a trifling hemorrhage.

When the hemorrhage is very profuse the blood will rush into the throat, exciting gagging, so that, for the moment, you might hesitate whether it was not discharging itself from the stomach. But if you wait until it has comparatively ceased, then you will easily perceive that it is, in fact, expectorated by coughing. On the other hand, when the hemorrhage is slight, it sometimes passes up through the air-passages into the throat, without exciting any cough. These peculiarities in the mode of expectorating blood should be carefully remembered, otherwise you may be easily deceived. Patients frequently try to conceal from themselves, and of course from their physician, the true source of the hemorrhage. They are always ready to refer its source to the throat, or to the nose, or to the stomach; and physicians sometimes take their statements as granted.

Whenever I am called to a patient who has had hæmoptysis, I always mark him as, probably, a tuberculous case. I have already alluded to the rare, exceptional cases. Cases of simple pulmonary congestion followed by hemorrhage, and by a return of the lung to a healthy condition, have never occurred to me. Neither have I ever met with a case of vicarious menstruation occurring as hæmoptysis. In acute bronchitis you will find a few streaks of blood, in pneumonia you will have the rusty expectoration. These are common cases, and the quantity of blood is too small to constitute a hemorrhage. You may ask, How little blood will constitute a hemorrhage from the lungs? I answer, if the patient has expectorated a teaspoonful of blood it is a hemorrhage; or even half this quantity, and mixed with mucus. A person coughs and expectorates a little mucus, deeply stained with blood. From time to time, he expectorates a little more blood in the same manner, without effort, without

pain; this is a trifling hemorrhage from the lungs. A copious hemorrhage would naturally excite more serious apprehension on the part of the patient; but a trifling hemorrhage, especially if repeated from time to time, is, perhaps, quite as diagnostic of tuberculous disease of the lungs.

Hemorrhage, although so important as a diagnostic sign of tubercles, very seldom proves fatal in its immediate effects; neither does it seem, as a general rule, to act unfavorably on the general progress of the disease. On the contrary, statistical tables prove, that those phthisical patients who experience hemorrhage, usually live longer than those who do not. Oftentimes the flow of blood is attended with a feeling of decided relief, especially if it assumes the characters of a passive hemorrhage. Sometimes, when it assumes an active character, attended with febrile excitement, and induces a condition of lung analogous to inflammation, it may produce injurious effects—an active period in the progress of the disease seeming to coincide with its occurrence. Thus hemorrhage is not always to be regarded in the same light, when you look at individual cases. But when you look at this symptom in the mass of cases, its existence must be regarded as exerting rather a beneficial influence than otherwise. There are many persons, more or less tuberculous, who, from time to time, expectorate even large quantities of blood, who after a little rest, to recruit the exhausted strength, return to their occupations, and live on, year after year, without any apparent loss of health. The most protracted case of phthisis I have ever known, lasting thirty-five years, was marked by occasional returns of, sometimes, very copious hemorrhage during this long period.

I have stated to you that hemorrhage from the lungs did not mark the stage of the disease. It may occur early or late in its progress. Formerly, hemorrhages were attributed to two causes, exhalation from a free surface, as the mucous membrane, or rupture of a blood-vessel. But microscopic observations have established the fact that there can be no such thing as an exhalation of blood, it must always escape from a ruptured vessel. It may take place from numerous capillary vessels ramifying upon the surface of the bronchial mucous membrane, and this is prob

ably the fact in a great majority of the cases of hemorrhage connected with tubercles—always, indeed, when it occurs at an early stage of the disease. Its mechanism is simply this: The tuberculous deposit, by pressing upon the capillary vessels of the lungs, obstructs some of them, while others become congested in consequence. These congested vessels, when seated in a mucous tissue, become ruptured from distension, and discharge blood. There is no reason to believe that the capillaries of the air-cells or of the common cellular tissue of the lungs are ruptured. If they were ruptured, you should find pulmonary apoplexy in fatal cases. But this is not the case. Hemorrhage may, indeed, occur from a ruptured vessel of considerable size, from ulceration. This can only happen in an advanced stage of the disease; even then it rarely occurs from this cause. When it does occur, it is usually very abundant and difficult to control.

When patients are questioned as to what may have excited the hemorrhage, they can seldom state any thing which seems likely to have acted as an exciting cause. In a large proportion of cases it occurs quite unexpectedly, without premonitory symptoms. Sometimes an unusual effort, especially of the chest, seems to act as an exciting cause; and in women, the occurrence of the menstrual period may induce the same result.

Hemorrhage would undoubtedly occur more frequently and copiously than it does in the progress of phthisis, and as the lungs become filled with the tuberculous deposit, was it not that the quantity of blood circulating in the lungs is materially diminished. The blood emaciates like the other parts of the human system. This influence is felt also by the heart, which does not increase in size with the progress of the pulmonary obstruction, as you might suppose, but it rather diminishes in size, with the diminished quantity of blood in the circulation.

I am not afraid that I have dwelt too long on this important symptom. Its frequency, its diagnostic value, its influence on the prognosis, the great alarm it usually excites in the patient, and in the family, make it worthy of the most careful consideration.

Dyspnœa is a constant symptom of phthisis, and it usually occurs very early in the disease, sometimes even before the

cough. It does not generally reach the degree you will notice in heart disease, or in emphysema of the lungs. Sometimes, however, it is a very distressing symptom. It occurs most decidedly in cases in which a large crop of tubercles are suddenly deposited in the lung, or in cases of tuberculous infiltration. In advanced cases of the disease, it usually increases in degree, but less decidedly than you might expect from the known condition of the lungs; this is probably owing to the diminished quantity of the blood. It is aggravated temporarily by congestion of the lungs, with or without hæmoptysis, by the accumulation of mucus in the bronchi, by pleuritic pain, by pneumothorax, and especially by active exercise. When a patient is suddenly attacked with increased dyspnœa, you should always examine the lungs carefully. You may discover an increased secretion into the bronchi, or the physical signs of pneumothorax. Sometimes a careful examination of the lungs discovers no cause of even an intense dyspnœa, which has suddenly supervened. You should then examine the heart. A blood polypus may have formed in the right cavities of the organ, and thus obstructed the circulation. I have met with several such cases.

Pain in the chest is almost always found to exist in this disease. Some patients are, indeed, nearly or quite free from it, other patients suffer more from it. Generally, the development of pain is a guide to the seat of the disease. The side first and principally affected, points to the lung first and principally diseased. This symptom has evidently two sources: first, the external intercostal nerves; second, the inflamed pleura. The first source of pain, is by far the most common. The flying pains, the external soreness of the chest, must be explained in this way. Frequently, pressure upon the spine between the shoulders, reveals much tenderness. Pleuritic pains are more fixed in their seat, they are apt also to be attended by increased febrile excitement. Neuralgic pains in the chest are not necessarily connected with tubercles. I make this remark, because, in certain cases, they might pass for more than their real value. Many persons whose health is delicate, and whose chests are simply weak, without being actually diseased, are liable to these pains. I have mentioned, also, that these pains

will usually call your attention to the seat of the disease, to the lung affected, when they exist in connection with tubercles. It is a good habit to examine the chest carefully for the physical evidences of disease, before you inquire as to the existence of pain. Doubtful physical signs may sometimes be confirmed or weakened by this method.

A difficulty of lying on the side most affected is a very frequent, but not a constant symptom. This fact will sometimes aid you in determining which lung is most at fault. It may be owing to external tenderness in the chest, but it is generally, I think, dependent upon the compression of the lung itself, creating general uneasiness, and an increased tendency to cough. In the latter stage of the disease, when an abscess exists in the lung, this fact is sometimes reversed. The patient can rest most quietly on the affected side. This position allows the secretions of the abscess to accumulate, for a time, in the cavity. If the patient moves to the opposite side, the contents of the abscess may flow out into the bronchi and excite coughing.

The constitutional symptoms of phthisis are generally very distinctly marked. Emaciation is the most prominent of these symptoms. It sometimes precedes all the other symptoms. A patient loses flesh for some unknown reason, and by-and-by the symptoms of tuberculous disease appear. Emaciation is associated with a certain degree of anemia, and their combined influences are often early noticed in the countenance. There is a peculiar physiognomy, even in the early stage of the disease, especially in the thin and pallid cheek, and in the bright eye. As the disease advances, this symptom increases, until at length it may reach an extreme degree. Every organ in the body seems to emaciate, except the liver and the heart, not merely by the absorption of fat, but by a wasting of all the component tissues. The blood even may be said to emaciate.

Emaciation is far from being always a progressive symptom. Patients, especially in the more chronic cases, often improve in flesh, gain rapidly, sometimes many pounds in weight, and then lose it again. These changes may continue through a series of years, and after many years, the patient may, on the whole, keep his usual weight. But it seldom or never happens, I think, that

a patient who has once emaciated from the tuberculous disease ever regains entirely his original weight, that is, his usual weight before the attack; and it more commonly happens, even in the most favorable cases, that there is, on the whole, a gradual loss. These temporary changes indicate the activity or the indolence of the lung disease. Sometimes other causes, besides the progress of the disease, induce emaciation, especially loss of appetite and diarrhœa. On the other hand, you will sometimes see patients actually gain flesh under favorable circumstances, especially such as improve digestion, while the pulmonary disease has lost nothing of its activity. You must not trust to the statement of patients, when this important symptom is but moderate in degree, nor even to their friends. Examine the body, especially the arms, and see if the skin is more loose than it should be, or trust to a pair of accurate scales.

Loss of strength attends emaciation, and follows the changes that it undergoes.

The pulse is, almost universally, accelerated. I have occasionally met with a patient, who unquestionably had phthisis, whose pulse was not above the natural standard. Sometimes I have ascertained that the pulse was naturally below the standard, and I suspect that this will generally be found to be the case in such instances. In the early stage, the excitability of the pulse is often its most striking characteristic. When the patient is tranquil, the pulse is tranquil, but the least excitement carries it up ten, perhaps twenty, strokes in a minute. If you compare the morning with the evening pulse, you will often find a difference, partly perhaps from the excitement of the day, partly from the natural tendency to acceleration which seems to exist in the evening. When the disease has taken a strong hold, the pulse indicates it. It is permanently and decidedly accelerated, seldom less than one hundred in the minute, and the stroke is sharp and active. An improvement in the symptoms generally, does not always bring with it an improvement in the pulse. It is a most obstinate symptom, this accelerated, quick pulse. This and the cough are the last symptoms which disappear in cases which recover. In the advanced stage of the disease, of course the acceleration of the pulse becomes more and more decided.

Hectic symptoms—irregular morning chills, evening febrile excitement, night-sweats—sometimes occur in the early stage of the disease, from simple irritation produced by the tuberculous deposit. But more frequently you will notice these symptoms when the tubercles begin to soften, and especially when abscesses have formed. Temporary causes, a pulmonary congestion or inflammation, gastric derangement, mental or physical excitement, will aggravate these symptoms, and perhaps cause their irregular appearance. A tendency to chills is sometimes shown by an increased sensibility to cold, and the evening exacerbation appears only in an increased warmth of the palms of the hand and of the soles of the feet. The countenance is apt to become more animated, the eye to brighten, and, in delicate complexions, the fine blush of excitement gives a new beauty to the features. In the morning, the pale cheek, the languid expression, point again more clearly to the ravages of the disease. In the advanced cases, you will often notice a small, circumscribed redness in one or in both cheeks.

This evening excitement terminates in its natural crisis, nocturnal perspiration—sometimes slight and limited, at other times profuse and general. The night-sweats generally keep pace with the febrile excitement, and if any temporary cause increases the latter, the former increase also. Chills are frequently wanting, and the patient may be ignorant of any evening excitement, although there is sweating at night. A leading characteristic of hectic is its irregularity. It disappears without apparent cause, or from the use of certain remedies which do not exert any influence on the disease itself.

Hectic symptoms do not necessarily indicate the existence of tubercles, or, indeed, of any fixed disease. Feeble persons, if any temporary derangement of the system occurs, are apt to have night-sweats.

If a patient is affected with a slight deposit of tubercles, or has them in an inactive state, no hectic symptoms exist. But if he happens to contract a pneumonia, or other inflammatory affection of the chest, night-sweats are apt to supervene. The marked existence of this symptom in an acute inflammatory attack may lead you to the first suspicion of tubercles.

There is no chronic disease that I am acquainted with, in which the mind is so singularly affected as in phthisis. The hopefulness, the buoyancy of spirits, are, indeed, remarkable. The least improvement in the symptoms is at once hailed as the harbinger of returning health, while their aggravation is explained by a thousand trivial excuses. It is difficult to know, sometimes, whether the light and trivial way in which patients speak of their symptoms is to be regarded as an effort to conceal a fatal truth, or the result of real indifference to their condition. In cases in which there is a strong family predisposition to the disease, and if other members of the family have already died, the reappearance of the disease often excites great alarm in the family, and the patient, forewarned of his situation, is really anxious, but attempts to present his case in the most favorable light. Occasionally, also, when the patient is dyspeptic, he experiences a good deal of alarm, and is apt to take a gloomy view of his situation.

This almost universally cheerful, hopeful state of mind, is an indication that a dyspeptic condition is not often a cause, or a concomitant of the early stage of phthisis.

Patients with phthisis generally suffer very little from cerebral symptoms at any stage of the disease. Sometimes a little headache is present, hardly any thing else. But occasionally you will see patients, especially children, who complain of an intense and constant headache, and perhaps, at last, attended with paralysis, or with convulsions. In these cases, rare in adults, you must look for disease in the brain, and after death you will probably find tubercles in the substance of the organ, or in the membranes.

Those who maintain the opinion that imperfect digestion and the mal-assimilation of the food is a leading cause of the tuberculous deposit, have failed, I think, to establish the fact. The symptoms of dyspepsia, although they may precede and attend the early development of the disease, are by no means of very frequent occurrence. You will constantly meet with cases in which the digestive powers possess the average degree of healthy action, until in the progress of the case they begin to fail from the general weakness which the disease produces. This is what you must expect in all chronic diseases, especially in those in

which debility is a prominent symptom. The powers of the stomach are weakened, the appetite is impaired, the food produces oppression, flatulence, and if the attempt is made to tax too much the weakened organ, vomiting will ensue, and very likely a low inflammatory action will be established, which it will be very difficult to control. You will not unfrequently find the evidences of this action after death. The mucous membrane of the stomach is thickened, mammillated, softened—sometimes injected, at other times presenting the grayish tint of chronic inflammation. Vomiting not unfrequently occurs without any special derangement of the stomach, after violent fits of coughing. These cases should be carefully distinguished from those in which the stomach is primarily at fault. When the patient complains of tenderness and of heat in the epigastric region, when he vomits frequently bile and mucus, there is reason to believe that inflammation of the mucous membrane of the stomach exists. Ulceration of this organ is very rarely met with.

It is very common also for patients in the advanced stage of phthisis to experience some diarrhœa. Indeed, few escape this symptom altogether. At first you will notice that the bowels are moved by slight causes, by a little irregularity in living, for instance, or that the daily motions are rather free and loose without positive diarrhœa. If this disposition is not held in check, the symptom becomes more decided, and the free condition of the bowels becomes one of the most prominent features of the case. An uncontrollable diarrhœa may supervene, which will hasten materially the fatal issue, and after death you will find ulceration of the mucous membrane, tuberculous deposits, thickening and softening of the mucous membrane, enlarged mucous follicles, especially near the termination of the ileum and in the colon. Much of the ulceration in these cases is undoubtedly dependent on the softening of tuberculous matter deposited among the coats of the intestines.

I have mentioned the condition of the digestive organs, although I am now speaking to you of simple phthisis, because, in the simplest cases, these organs are more or less affected before the end of life. When inflammation, and especially ulcera-

tion, occur, with their aggravated symptoms, then the condition of these organs becomes, properly, a complication which, in fact, may be more prominently marked than the original disease. But the change from slight functional disturbance to confirmed organic disease is often gradual, imperceptible, and many cases do not advance beyond the first condition. It is on this account that I place before you an outline of the whole subject at this time. I shall have occasion in another lecture to return to this subject while speaking of the complications of phthisis.

The same thing is true in relation to the laryngeal affection in phthisis. Most patients complain, at times, of a slight hoarseness, especially in the evening, with some degree of irritation, accompanied by a chronic inflammation of the fauces. This condition is often trifling, but it is seldom absent entirely. It not unfrequently passes into a much more serious disease, ulceration of the larynx, and is attended by most distressing symptoms—as permanent loss of voice, pain in the larynx, difficulty in swallowing. I have already discussed this subject so fully in my lectures on laryngitis, that I need not now speak of it more in detail.

The condition of the menstrual function in females, in this disease, is an important consideration. Many young females cease to menstruate, they become pale and feeble, they emaciate somewhat, and the whole attention is concentrated upon the cessation of this menstrual function. Active emmenagogues are prescribed, but without effect, and gradually the symptoms of phthisis become so evident as no longer to be mistaken. Some physicians have thought that the disorder arising from the menstrual suppression might lead to the deposit of tubercles in the lungs. The reverse of this is much nearer the truth. Suppression of the menses is sometimes one of the first, perhaps the very first prominent symptom of phthisis, and a careful scrutiny of the case will often discover this to be the fact. Examine and ascertain if there is not a trifling cough, or other pectoral symptoms, and especially if there are not some of the physical signs of incipient phthisis.

As a general rule, the menstrual function continues during

the early stage of the disease, the quantity of the discharge gradually diminishing, or ceasing abruptly about the time that the tuberculous deposit begins to soften. Now and then, you will meet with cases in which the function continues during the whole progress of the case. Females are so liable to the cessation of this function from a variety of causes, and its existence is so variable in the course of early phthisis, that it generally possesses but little value in the diagnosis of the disease.

LECTURE XVII.

TUBERCLES IN THE LUNGS.

Tuberculous phthisis.—Appreciation of the physical signs.—Tuberculous infiltration; its diagnosis from pneumonia; from pleurisy.—Latent phthisis.—Chronic phthisis.—Symptoms that attend the curative process.—Complications: chronic laryngitis; tubercles in the brain; chronic peritonitis; hydro-pneumothorax; fatty liver and heart.

The physical diagnosis of phthisis is capable of affording you invaluable assistance, especially in the early stage of the disease. Cases, not unfrequently, present themselves, about which a good deal of uncertainty exists, when you attempt to judge them by the rational and the constitutional symptoms only. The cough has assumed the characters of that accompanying simple bronchitis, if you can trust to the description of the patient; there has been no hæmoptysis, and no marked emaciation or acceleration of the pulse is noticed. In these doubtful cases, a careful physical examination of the chest will frequently remove all uncertainty. It is true that cases occur in which you will not be able to detect any physical signs of the existence of tubercles, even when they exist. This happens, particularly, when the deposit is disseminated through the pulmonary tissues. I have known a case in which the lungs were found, after death, studded with minute miliary tubercles, in which no physical sign of their existence could be detected. A fact like this should

never be forgotten. A much less extensive deposit of scattered miliary tubercles may exist, and produce suspicious symptoms, while the physical signs are entirely inappreciable. Generally, however, with great care, if the tuberculous deposit exists in clusters, as it usually does, you will detect the disease by its physical signs.

There is no case of pulmonary disease in which the physical examination should be conducted with more precaution. If you make a careless examination, while the patient is sitting or lying and with chest more or less covered by clothing, you will frequently make mistakes, which might be avoided. In all such cases, the upper portion of the chest should be exposed, if possible, or at most be covered by the thinnest and most pliable material, as, for instance, by a thin linen handkerchief. The patient should be placed standing, and resting the shoulders against the side of the room. He should rest equally upon both feet, and stand in an easy position. You should then percuss under each clavicle, gently, equally. A slight difference in the sound will usually be detected. A slight degree of dulness exists on one side, which diminishes as you percuss a little lower down in the chest. You may find a perceptible dulness on the first rib, while you obtain a perfectly clear and natural resonance on the third rib. It not unfrequently happens that the dulness is more distinctly recognized when you percuss directly upon the clavicle. It may happen also that both lungs have become equally affected at the same time; but that is a very rare circumstance indeed. This, of course, will render the diagnosis less clear, as you will lose the great advantage of comparison. Yet an experienced percussor will notice that the sound on both sides is less resonant than natural directly under the clavicles, while below, as, for instance, upon the third rib, it is more clear. Posteriorly, no advantage can be derived from percussion; the muscles are too thick about the shoulders to admit of any decided advantage, even in cases in which the pulmonary condensation is much greater than in incipient phthisis.

As the tuberculous deposit increases, the dulness on percussion also increases, and at length becomes quite decided. If a cavity forms in the lung of a considerable size, and is superficial, the

resonance on percussion becomes again more clear, but still without the natural clearness of healthy lung. It resembles, in some degree, the sound produced by percussing over a hollow organ, as, for instance, over the stomach; and frequently it gives a chinking sound, which the French have called the cracked-pot sound. This cracked-pot sound, however, is not, as some have supposed, a sure indication of a cavity in the lung. I have perceived it where no cavity existed, especially where the chest was unusually sonorous, and contained much mucous secretion.

When you percuss a patient carefully, using the left fore-finger as a pleximeter, you will get another indication of pulmonary condensation besides the dulness. You will perceive a want of that elasticity which the parts naturally exhibit. This is an important fact in connection with the dulness, and it should be carefully noticed. In addition to this, I may say, that when the disease has made some progress, the clavicle of the side most affected seems to be more prominent than that of the other side, from a diminution in the actual size of the lung, from pleuritic adhesions at that spot, and perhaps, also, from a certain degree of local emaciation having occurred. In advanced cases of the disease, this flattening of the chest under the clavicle is often very marked.

The most important early change in the respiratory murmur in incipient phthisis is feebleness, sometimes united with harshness, or with a jerking inspiration. Sometimes you will notice, listening with attention, a kind of dry crackle, repeated, perhaps, once or twice at the end of inspiration. Generally, the murmur of expiration is prolonged, and more distinctly on the right side, with the same amount of disease, than on the left side, for the reasons I have stated in my introductory lecture. These changes are observed at the summit of the chest, and there only; sometimes, they are most distinct under the clavicle; sometimes behind, above the spine of the scapula. Indeed, I may remark that the changes in the respiratory murmur are often more clearly appreciated behind than in front. It is important to remember this. You must remember, also, the great advantage of comparative auscultation, of comparing carefully the same spots in each lung; for the respiratory murmur, like the reso-

nance on percussion, becomes much more valuable in the diagnosis of doubtful cases when this is carefully attended to; never forgetting, however, that the expiratory murmur is naturally more distinct and prolonged at the summit of the right lung than at the summit of the left lung.

With the increased growth of the tuberculous deposit, aided by new deposits, the condensation of the lung may become so complete that hardly any respiratory sound can be distinguished at the seat of the disease. But this is rare. The condensation, however great, usually leaves the larger bronchi of the part open, and a bronchial respiration is perceived, not often, however, of that strongly marked character you will notice in pneumonia of the apex of the lung. At the same time, you will notice at the seat of the disease a mucous rattle, which does not extend usually beyond the limit of the disease, or if it does, it becomes much less distinct. This mucous rattle, limited to the upper portion of the chest, is a most valuable fact in the diagnosis of phthisis, at a somewhat advanced period of its progress—the period when the tubercles have commenced to soften. It is not necessarily connected with a bronchial respiration, or with dulness on percussion. In cases in which the tubercles are not numerous and scattered, these signs, as I have already told you, may fail; but when these same tubercles soften, the irritation they create may be disclosed by the mucous rattle from increased bronchial secretion. Such a case as this will now and then present itself—the dulness on percussion, the alteration in the respiratory murmur, are very slight, if they exist at all—you have nothing satisfactory at your first examination. But the next day, especially if you make an early examination, before the mucous secretion has been expectorated by the morning cough, you may detect a mucous rattle under the clavicle, or above the spine of the scapula, and nowhere else. This is a very decisive fact. This mucous rattle is not necessarily very distinct; it is, on the contrary, sometimes rare and indistinct, a few bubbles of air breaking, especially during inspiration. In other cases it is distinct and abundant, and gradually changes into a gurgling sound, as the tuberculous matter becomes more and more softened, until, finally, cavities are formed.

The cavernous respiration, sometimes dry, sometimes accompanied, and, more or less, masked by a gurgling, is not always heard during life, although cavities are proved to have existed by post-mortem examination. Indeed, I think this is frequently the case. If the abscess has not opened into a bronchus, of course it can give no sure sign of its existence. Or if it has, the communication may be small and indirect, so that the air will not enter it freely and generate a cavernous sound; or, finally, the abscess may be small and deep seated in the lung. Sometimes the peculiar resonance on percussion may induce you to suspect that a cavity exists in the lung, when the sound of respiration does not indicate it.

Increased resonance of the voice, bronchophony, and sometimes pectoriloquy, are developed at the summit of the affected lung. You must not forget that there is naturally rather more resonance of the voice at the summit of the right lung, than at the summit of the left lung.

I have described the phenomena of simple phthisis, which may be regarded as the type of the disease. Let me now call your attention to some other forms of the disease.

An *infiltration of tuberculous matter* into the pulmonary tissues, sometimes occurs as the primary lesion in the place of the miliary granulations. It is not to be distinguished from the latter form of the disease, except by the physical signs. The infiltration is usually rapid, extensive, and affects frequently the lower and posterior portion of one lung. Hence you may expect to find many of the physical signs of pneumonia, with which, in its acute form, the disease is apt to be confounded. There is marked dulness on percussion, extending perhaps from the base of the lung to the lower angle of the scapula, but not usually passing a vertical line drawn from the axilla downward. The respiratory sounds may be simply feeble and indistinct, but generally you will hear a bronchial respiration; and as I think, possessing, sometimes at least, peculiar characters which distinguish it from the ordinary bronchial respiration of pneumonia. It gives the impression of air passing through fine tubes, and as if drawn through them by a sort of suction. In the early stage it is dry, but as the tuberculous infiltration softens, a mu-

cous rattle is gradually established; and finally, when cavities form, a gurgling of mucus may occur, or even without their formation. In its early stage, especially if the infiltration is very great in degree, it might be confounded with pleurisy. The marked dulness, the absence of respiratory sound which may occur in this case, would lead you to suspect a pleuritic effusion. But these physical changes will not probably pass the median line drawn downward from the axilla, as they would do, probably, in a case of pleuritic effusion. Neither will you detect egophony. You may, indeed, have your doubts, even after a careful examination; then wait a little before deciding. The future progress of the case will satisfy these doubts. Besides, in these cases, the rational and constitutional symptoms are those of phthisis, not those of pneumonia, or of pleurisy. The attack may be acute—indeed, it generally is so; but still the fever is moderate, the pain in the chest trifling or absent, and there is no characteristic expectoration. Consider also the general condition of the patient. Has he the aspect of phthisis? Is there an unusual acceleration of the pulse? Is there a tendency to night-sweats, and, above all, has hæmoptysis occurred?

When tuberculous infiltration affects the base of the lung, the upper portion of the side affected, as well as the opposite lung, may present no indications of disease. But watch the case. By-and-by, you will find the physical signs of phthisis presenting themselves at the apex of one, or of both lungs; and thus any previous doubts as to the nature of the case will be entirely removed.

Latent phthisis is another form of the disease that you should thoroughly understand. You must know, that an extensive deposit of tubercles may occur in the lungs, and the process of softening even, be well advanced, without a single rational symptom of the disease—no cough, no pain, no dyspnœa. Such a case happened to me many years ago, and since that period I have met with other cases. What is the reason of this? Is it because the disease exists in great activity in some other organ? in the peritoneum? in the intestinal canal? The disease in the lungs is masked by disease in some other organ. But the constitu-

tional symptoms of phthisis exist. This should excite your suspicions, and lead you to a careful examination of the chest. Thus you may find the physical signs of phthisis well developed, or obscure and indistinct, or even absent. Still, keep your attention directed to the chest. Examine it carefully from time to time, and, sooner or later, the true character of the disease will be revealed. These remarks apply with particular force to the cases of children. In them the disease of the lung is sometimes developed secondarily, after other organs have become affected, and its true nature may only be discovered after the lungs are found to present the physical signs of phthisis. In adults, the lungs are probably affected as soon, at least, as any other organ; but still, by the overpowering activity of the disease in remote parts, it may continue latent.

It is in this way, I suppose, that pregnancy operates upon phthisis. There is a common impression, that pregnancy retards the progress of phthisis; probably it only renders it latent, and thus an apparent rather than a real advantage is gained. That it produces neither of these results, in some cases, I am very well convinced, and the practitioner who recommends it to his patient may be disappointed even in a temporary advantage. Even supposing that the progress of tubercles is retarded during the existence of pregnancy—what is the final result? As soon as delivery has taken place, the pulmonary disease usually advances with great rapidity, and, in addition, a child with a strong tuberculous tendency is born. Certainly there is no great advantage in these results, and you will, I hope, be disposed to adopt the opinion that I have formed—never to advise pregnancy to a tuberculous female. Cases of the kind will occur often enough, and the evil consequences be experienced, without, or in opposition to, your advice.

There are cases of phthisis which may be called *chronic* or *intermittent*. That is, the symptoms commence and progress to a certain point, and then an improvement takes place, perhaps a very decided improvement. The patient may think himself nearly recovered, when the symptoms return again, and continue for a time, and again improve. These changes for the better, seldom carry the patient to the point of health he enjoyed before

the last attack. There is a little falling off; the disease is, in fact, progressing insidiously, until, at length, the powers of the constitution are fairly undermined. These intervals of improvement sometimes commence with the expectoration of the chalky deposit in the lungs, an indication that the tuberculous matter is undergoing a transformation favorable in its nature. At other times, the improvement may coincide with the discharge and cicatrization of a tuberculous abscess, or, perhaps, there may be only a cessation in the development of the tuberculous matter. With these conditions, you will find, after death, in persons who did not at the time experience any active symptoms of tubercles—chalky concretions, cicatrices, cavities nearly empty and lined by a fibrous membrane, or simply tubercles, with a dark withered appearance, and without surrounding congestion or inflammation.

The aggravation of the disease in this class of cases is, no doubt, generally owing to a new deposit of tubercles. For you will find, after death, limited traces of old and imperfectly cured tuberculous disease, with a more or less abundant deposit of recent tubercles in various stages of their progress. Or, on the other hand, the tuberculous deposit which had been arrested in its development, has become active again, and passed on to more serious changes.

Some of these cases undoubtedly terminate in a restoration to health, yet after a long struggle. The patient continues to cough a little, at least at times; perhaps he expectorates occasionally a chalky concretion. He sometimes has an uneasiness about the chest, and a little dyspnœa; his pulse is rather disposed to acceleration, his countenance is rather pale, and his strength and flesh moderate in degree. He cannot be called robust; but he is still able to follow a laborious occupation. As age advances, his condition probably improves, and you will find him, after many years, without any symptoms of pulmonary disease, and finally dying of some malady quite independent of the tuberculous diathesis. Yet when you examine his lungs after death, you find distinct evidences of former disease. Adhesions at the summit of the lung, puckering and linear depressions of the surface, near the apex, while internally you will notice a whitish

linear mass, with perhaps branches radiating from it, and one or more of these branches evidently terminating in a bronchus; or you will find even more distinct traces of a cavity, which is still partially open, is lined by an organized membrane, and containing perhaps a serous or a mucous fluid, or portions of old and dry tuberculous matter; or, finally, you will find chalky concretions.

I have briefly recapitulated some of the leading changes that mark the cure of tuberculous disease—an imperfect cure, perhaps, you will say—in order to be able to state to you more distinctly an important fact in connection with the physical condition of the lungs. As a lung once affected by the tuberculous deposit never regains entirely its primitive condition, the part affected remaining always less pervious to air, the parts destroyed not being restored, the lung being besides more or less condensed, you must expect, in such a condition as this, that some of the physical signs will also continue. You will not be surprised to learn that a certain degree of dulness under the clavicle will continue to exist for life, that the respiratory murmur at the summit of the lung never regains its natural softness and strength. It may remain feeble, harsh, jerking, or sometimes be more decidedly altered by a prolonged expiration, or even by a slight bronchial respiration. Indeed, there is no reason why a cavity in the lung, which does not cicatrize, but which remains open, and still communicates with a bronchus, may not continue for life to present the physical signs of a cavity.

The facts which I have just stated, should put you on your guard not to judge of a case entirely by the physical condition of the lungs, but to carefully compare this with the symptoms and previous history of the patient. Indeed, I must say again, what, I believe, I have often said before, that the practitioner who leans too exclusively upon any one of the three classes of indications, and especially upon the physical signs, will sometimes commit the gravest errors, which a more comprehensive inquiry would have prevented.

There are certain *complications of phthisis*, which enjoy a superior importance from the fact that they also are of a tuberculous origin, or evidences of the scrofulous diathesis. These

complications may occur after the disease in the lungs is well developed. But it sometimes happens, indeed not unfrequently, that the disease in other organs is the first in the order of development. It makes its appearance before tubercles in the lungs have revealed themselves with sufficient distinctness to be clearly recognized.

In a former lecture I have spoken of chronic laryngitis as usually connected with tuberculous disease in the lungs.

In speaking also of the condition of the cerebral functions, I have had occasion to point out the remarkable absence of cerebral symptoms in phthisis, at least in the adult. In children, cerebral symptoms are more common, and are so often unattended by distinct chest symptoms, that until of late years their true connection was not ascertained.

When, in the course of phthisis, the patient complains of constant and severe pain in the head, especially in the forehead, followed by vomiting, by prostration of strength, agitation, alternating with stupor, convulsive movements, paralysis, and coma, and the case passes on to a fatal issue, often with an apparent diminution of the chest symptoms, you may expect to find after death tubercles in the brain. Usually this deposit will be found in the membranes, and especially in the pia mater, at the base of the brain. Sometimes tuberculous masses are found in the substance of the brain, and you will notice in these cases accompanying marks of inflammation—congestion of the vessels, adhesions, softening of the cerebral substance, especially of those parts which enter into the structure of the ventricles—the septum lucidum, the fornix, &c. Frequently a considerable effusion into the cavity of the ventricles, sometimes of transparent, sometimes of turbid serum, will be noticed.

This disease, when it attacks the membranes of the brain in children, has long been called acute hydrocephalus, its real nature being quite overlooked. The same affection also exists sometimes in the adult, and is not distinguished by any peculiar symptoms.

I am not aware that the deposit of tubercles in the substance of the brain can be distinguished from the same deposit in the membranes by any certain, well-defined symptoms. The former

is no doubt generally a much more acute affection, terminating usually in less than three weeks; while the latter is commonly a chronic disease, lasting often for months. Indeed, many of the cases of what is called chronic hydrocephalus are connected with a tuberculous deposit in the substance of the brain.

Chronic peritonitis is another of the diseases of tuberculous origin; and, like tubercles in the brain, it often develops its symptoms before the lungs are apparently affected. Like the cerebral disease, also, it tends to render the chest disease latent. When a patient is attacked with pain in the abdomen, not usually severe and fixed, but wandering, moderate in degree, often transient, and this is followed by a tympanitic swelling from gas in the intestines, and gradually by fluctuation; when, after a time, the distension diminishes, from an absorption of the liquid effusion, and from a partial removal of the gas, and the abdomen remains enlarged with a dough-like feeling, or with firm ridges, giving the sensation of an unequal firmness on pressure; when, in addition to these local symptoms, the constitutional symptoms of tuberculous disease are present—emaciation and loss of strength, hectic, accelerated pulse, and especially, if the rational or physical signs of tuberculous disease of the lungs exist —if all these conditions are present, you need have no hesitation in referring the abdominal symptoms to a tuberculous peritonitis. After death, you will find your diagnosis confirmed by an abundant deposit of tubercles in the peritoneal cavity—false membranes loaded with tubercles and gluing the folds of the intestines together. Sometimes you will find tubercles under the peritoneum, and perhaps a considerable effusion of serous, sero-purulent, or even purulent matter, in the cavity of the abdomen.

I may remark, that acute peritonitis sometimes occurs in connection with phthisis. It may be dependent upon perforation of the intestine, ordinarily at the terminal portion of the ileum, and probably also from a tuberculous ulcer. The symptoms in these cases are often obscure, but always rapid. A sudden prostration, a very rapid pulse, a tumid, tympanitic abdomen, with or without pain and tenderness, and speedy collapse and death, are the usual symptoms.

It may happen, that a tuberculous abscess discharges its con-

tents into the cavity of the pleura, and, at the same time, a communication is established with the bronchi, so that air passes into the same cavity. The immediate effect of this accident is a compression of the lung, and usually, but not always, the rapid development of pleuritic inflammation. At first, it is the air alone which compresses the lung; but, in a great majority of cases, the inflammation excited produces its usual consequences—the rapid effusion of a serous fluid, which still further compresses the lung. In certain cases, indeed, this liquid effusion does not occur, at least in any considerable quantity, for several weeks, even, after the accident; but this is rare. When air alone exists in the cavity of the pleura, this complication is called *pneumothorax;* but when a liquid effusion also exists, it is called *hydropneumothorax.*

When you examine a case that has reached its fatal termination, you will find, on puncturing the cavity of the pleura, that a considerable quantity of gas will escape—sometimes inodorous, sometimes offensive; and on opening the chest, a large cavity is found, formed by the pleural sac partially filled with serum, often with a reddish tint, and rendered more or less opake by particles of lymph, or by pus globules. Sometimes, indeed, you will find a distinctly purulent fluid. The surface of the pleura is covered by layers of coagulable lymph, and the lung is compressed and driven from the walls of the chest, as far as the previously existing adhesions will permit. The upper portion of the organ is almost always found adherent to the chest, and sometimes more extensive adhesions exist. If now you pass a tube into the trachea, and attempt to inflate the lung, you will usually notice the escape of air from a certain point, and this will lead you to the spot where the perforation of the lung has taken place. You will find a small opening in the pleura passing into the lung and into a tuberculous cavity, in which one or more bronchi terminate. This tuberculous cavity may be large, but it is often very small. The lung itself is the seat of very considerable disease, or the tuberculous deposit may be very limited. Sometimes, after a careful search, you will not be able to find the point where the lung has been perforated. You may find a tuberculous cavity which has a communication with the pleural

sac, but none with the bronchi. Or you may not find even this, but only tubercles in the lungs, in various stages of their progress, and not an abscess opening into the pleural cavity. These circumstances have led to the opinion that hydro-pneumothorax might occur without perforation of the lung at all, or at least without any communication with the bronchi. It is supposed that gas may be secreted by the pleura, or that the products of a tuberculous abscess emptied into the pleural sac, or even the product of pleuritic inflammation, may be decomposed, and gas thus generated. But does the inability to find after death a communication between the bronchi and the pleural sac, in certain rare instances, compel you to believe that no such communication has ever existed? I think not. You must remember that the disease is almost invariably associated with tubercles in the lung, and with tubercles advanced in their progress. Now, it may readily happen that the opening into the pleural sac, especially if it be small, may become obliterated by a layer of the lymph which is so abundantly spread over the lung; or that the communication with the bronchial tubes may have been obliterated after the tuberculous matter in the abscess has been discharged, and the cavity itself has been partially obliterated, reduced to a mere sinus, by the surrounding pressure upon the lung. Both these conditions are likely to occur, and thus a communication with the external air which once existed, no longer exists.

The point where the rupture of the abscess into the pleural sac occurs is most frequently in the upper lobe. It is not at the summit of the lobe, for the adhesions which exist there usually prevent this, but lower down, opposite the angle of the third or fourth ribs. Commonly but one opening exists, but sometimes there are several openings discovered. The communication of the abscess with the bronchi may also be single, or by several openings, especially if the abscess is large.

You must not always expect to find a considerable liquid effusion in the chest in these cases, especially if the accident has proved rapidly fatal. You may find a little reddish serum in the pleural cavity, with lymph, or a moderate quantity of pus—a good deal of gas, and but little fluid.

I may remark, that hydro-pneumothorax sometimes occurs in

gangrene of the lung, and that it is produced by the same mechanism. A gangrenous eschar breaks into the pleural sac, and a communication is also formed with the bronchi. Although it is difficult to admit the accumulation of gas in the pleural sac from the decomposition of the matter of a tuberculous abscess, or from the secretions of the pleura, it is not difficult to conceive that an eschar falling from the lung into this cavity might generate a pneumothorax without bronchial communication.

It is also possible that an emphysematous vesicle in the lung may rupture the pleura covering it, and thus produce a pneumothorax. The same may happen, and more frequently, when the lung has been injured by external violence. But all these cases are rare. When you find pneumothorax and its consequences, you must look to the tuberculous deposit as its almost constant cause.

Patients attacked with pneumothorax will be found, among those who have suffered for a considerable time, usually for months, with the symptoms of phthisis. The case is progressing in the usual way—when all at once the patient is seized with a pleuritic pain in the side, usually well marked, sometimes less distinct, accompanied with great oppression of the respiration, anxiety, and increase of the febrile symptoms. Sometimes the attack resembles rather a severe shock to the system than an inflammatory attack. There is no complaint of pleuritic pain, but great oppression, inability to lie down, a tendency to fainting, a rapid pulse, with coldness of the extremities.

If the chest be examined, the cause of this sudden commotion becomes apparent. The side affected is found to be unusually resonant on percussion, perhaps it is dilated; yet the respiratory sounds are indistinct or absent, unless at the summit and at the root of the lung, where the adhesions and natural attachments of the lungs prevent the organ from being pushed from the parietes of the chest. This sudden development of resonance on percussion, with dilatation of the chest, and absence of respiratory sounds, can only occur from pneumothorax. But in addition, you may find at once evidences of perforation of the lung—amphoric respiration and metallic tinkling may exist. As you examine the case from day to day, you will find that the chest is

getting dull on percussion at the base of the lung, and this dulness increases until it extends over one-half, or more, of the affected side. But sometimes this dulness is slow in developing itself; several weeks even, may pass before it exists in a decided degree, and until it does exist the evidence of liquid effusion is not apparent. With this evidence of liquid effusion you will have another sign of its existence. If the effusion is considerable, and the patient, when sitting up, be shaken sharply by the shoulders, the ear being applied to the affected side, a splashing sound is heard, like that produced by shaking a barrel half filled with a liquid. The amphoric respiration, the amphoric resonance of the voice, the metallic tinkling may continue to exist, if they have already been noticed, or one, or all of these physical signs may be developed in the progress of the case. Sometimes you will detect the amphoric resonance of the voice alone. Sometimes you will hear distinctly the amphoric respiration, and sometimes it is the metallic tinkling, alone or united with the amphoric respiration or resonance.

The cause of the variation in these physical signs will be apparent when you recollect the circumstances of their production. Thus there may be a communication between the bronchi and the pleural sac and a large cavity formed there, containing air, and probably fluid, yet no direct evidence of this communication exists. For this communication may be small or partially closed, and then the force of the air entering the pleura will not be sufficient to produce an amphoric respiration, or even an amphoric resonance of the voice. Perhaps, if the patient should be made to cough, the vibrations of the air in the cavity might be strong enough to generate these sounds. If the communication also happens to be below the level of the liquid effusion, no amphoric respiration can occur, unless by a change of position you can change the level of the fluid, and thus bring the point of bronchial communication above this level. So with the metallic tinkling, you may hear it or not. If the bronchial communication is below the level of the fluid, and this communication be of considerable size, you will probably hear it, as the bubbles of air rise up and burst on the surface of the fluid. But if the communication be small, it may require a stronger

effort, a cough, for instance, to develop it. You may catch it, for a moment also, when the patient rises from the recumbent position, as a few drops of fluid, after adhering to the top of the pleural cavity, drop off, one after another, into the mass of fluid below.

The most common cause of the metallic tinkling has been supposed to be the breaking of air-bubbles coming from the perforated lung into the great air-chamber in the pleural sac, and produced by the passage of the air through a portion of the liquid effusion in that cavity during inspiration. This is one of the methods by which this sound is produced. But in this case, the orifice in the lung being necessarily below the level of the fluid in the pleural sac, amphoric respiration could not occur; and were this the only way in which metallic tinkling could be produced, it could never exist at the same moment with amphoric respiration, unless, indeed, in certain rare cases, where there was more than one perforation of the lung, one above, one below the level of the fluid. The two, indeed, might exist together for a moment, when any drops of fluid were falling from the top of the chest into the mass of fluid below, thus producing a transient metallic tinkling, after which the amphoric respiration would continue to be noticed alone. But you will not unfrequently find these two physical signs existing together permanently. The metallic tinkling is also noticed during expiration as well as during inspiration; it is sometimes restored, when absent, by coughing, while sometimes it disappears after coughing. It is evident that the passage of bubbles of air through the liquid effusion in the pleural sac, will not explain these phenomena. You must look, then, for another explanation of the cases in which the metallic tinkling exists permanently with the amphoric respiration, as well as of those cases in which it accompanies both inspiration and expiration. And above all, you must seek for another explanation of its occurrence in cases where it is heard without any perceptible liquid effusion into the pleural sac. Mr. Castelnau's views will explain these cases. The metallic tinkling may be caused by the bursting of air-bubbles in the tuberculous abscess itself, just at, or near the point of perforation, and the sound thus generated resounding in the large air-chamber formed

in the pleural sac, changes a rattle, which would otherwise be a mucous rattle, into a metallic tinkling. It is probable, also, that a mucous rattle transmitted in nearly the same manner from the bronchi, would produce the same result.

The progress of this complication varies very much in different cases. Sometimes it is fatal in twenty-four hours after the attack; frequently the patient lives for a month or two, and I have once known a patient to live for nine months after the symptoms of perforation had occurred. After the first shock is recovered from, the pain in the side subsides, the dyspnœa diminishes, and the condition of the patient seems to improve. But generally, it is evident that the fatal progress of the case is hastened, and that the result cannot be far distant. The symptoms of phthisis continue, following their usual course, until death ensues.

The physical signs of hydro-pneumothorax in its more advanced stage, do not differ materially from those I have already described. The dilatation of the affected side continues, perhaps it has increased, and is complicated with œdema, extending to the corresponding arm. The signs on percussion continue—dulness below, resonance above—while the amphoric respiration and the metallic tinkling, one or both of them, may be heard, constantly, or only at intervals, or finally they may cease altogether, if the perforation of the lung is closed by lymph, or in any other manner. Still, while air and fluid exist together in the pleural sac, and in considerable quantities, the splashing sound of succession will continue. Or if the fluid, continuing to increase, finally displaces the air, which may be absorbed, this sign of hydro-pneumothorax will also cease, and the case present the physical signs of pleurisy, with copious liquid effusion.

Some of the most prominent signs of pneumothorax may exist—the metallic tinkling, the amphoric respiration and resonance of the voice—in certain cases of phthisis, where a large and superficial abscess exists in the lung, united to the walls of the chest by adhesions, and freely communicating with a bronchus. Here all the conditions of a large cavity with elastic walls exist, and amphoric sounds are necessarily the consequence. These cases are readily enough distinguished from hydro-pneu-

mothorax by a little attention. In the first place, there is no sudden development of symptoms, as in hydro-pneumothorax, no severe pleuritic stitch, no sudden dyspnœa, although it must be confessed, that in hydro-pneumothorax these symptoms are not always very prominent, or if they have occurred, they may have been forgotten by the patient. But the physical signs alone will generally establish the diagnosis. In tuberculous abscess there is no dilatation of the affected side, no increased resonance on percussion over the superior portion of the lung, no particular dulness on percussion over the inferior portion of the lung; the respiratory murmur is still heard, modified by the tuberculous deposit over most portions of the lung, and especially at its base, and finally, there is no splashing sound on shaking the chest.

I have spoken to you more fully of hydro-pneumothorax than of any other of the complications of phthisis, because it belongs properly to the diseases of the lungs. I might add to the number of tuberculous complications—as they exist, for instance, in the bronchial glands, in the glands of the neck, especially in children, or in the mesenteric glands, constituting what is commonly known as tabes mesenterica, but this would carry me beyond the field of my present inquiry—the diseases of the chest. I would simply make one remark in relation to the lymphatic glands of the neck. The chronic enlargements of these glands in children, do not necessarily imply tuberculous disease of the lungs; but in adults the case is, I think, different. In the few cases of chronic enlargement of these glands in adults which have fallen under my notice, that is, where a chain of these glands were enlarged in the lower portion of the neck, I have always found the evidences of tubercles in the lungs, and particularly in the lung corresponding to the enlarged glands.*

* The lymphatic glands, situated at the root of the lungs, have long been known to be subject, in children especially, to considerable enlargement, from the deposit of tuberculous matter in their substance, and that, too, without much, or even any deposit of tubercles in the lungs. But it is to Rilliet and Barthez that we are indebted for a much more complete account of the symptoms they may cause, and especially of the physical signs they may produce or modify. According to these authors, the tumors produced by these glands, by pressing upon the superior cava, may cause œdema or lividity of the face, a dilatation of the veins of the neck, a hemorrhage into the cavity of the arachnoid; or by compressing the pulmonary vessels

It is a very curious fact, that while the body generally is emaciating to the last degree in tuberculous phthisis, that two organs, the liver and the heart, should be subject to a contrary influence—the intimate structure of the liver undergoing a fatty degeneration, while the surface of the heart becomes loaded with fat.

The *fatty degeneration of the liver* is a remarkable fact in the pathological history of phthisis. It is almost peculiar to tuberculous phthisis. It is much more common in females than in males. The organ is usually enlarged, and sometimes very much so. Its texture is more or less softened; it is pale, with a yellowish tint. Generally, you can detect the existence of fat, in a rude way, by making a section of the liver and observing the fat upon the blade or the scalpel; or, which is a better mode, a small portion of the organ may be placed upon tissue paper, and a moderate degree of heat applied, when you will discover the stain of the melting fat spreading upon the paper; or, a portion of the organ may be placed, for a time, in ether, which, by evaporation, will disclose the existence of fat which it has held in solution. But, what is a still better mode, you can readily detect the existence of fat by the microscope.*

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they may induce hæmoptysis or pulmonary œdema. Pressing upon the œsophagus, they may induce dysphagia, or upon the pneumo-gastric nerve they may render the voice hoarse or extinct, they may induce a spasmodic cough, or paroxysms resembling asthma. Compressing the air-tubes, they may cause a grave, sonorous rhonchus, or a feeble respiratory murmur. Or, pressing upon a bronchus at one point, and upon the walls of the chest at the opposite point, and thus establishing a direct, solid communication between the bronchus and the ear of the auscultator, a bronchial, or if the tube is very large, a cavernous respiration may be heard, *without any disease whatever* of the lung itself. And again, if disease does exist in the lung, this direct and solid medium of communication between the diseased portion of lung and the ear may exaggerate remarkably the physical signs of the pulmonary disease. A leading feature in these cases, as in other cases in which tumors compress the organs in the chest, is the intermittence or irregularity in the symptoms and signs they produce at different times of examination. These glandular tumors not unfrequently open a communication between themselves and the compressed bronchus, and the same accident has happened to the œsophagus and to the pulmonary artery. They may also communicate with a tuberculous pulmonary abscess.

* According to Lebert, the fat is first deposited in the cells of the liver. But, after a time, it becomes so abundant that the cells are destroyed, and the organ seems to be composed entirely of blood-vessels, biliary ducts, and fatty tissue. The

LECTURE XVIII.

TUBERCLES IN THE LUNGS.

Duration and prognosis of tuberculous phthisis.—The possibility of its cure.—Diagnosis.—In the early stage it may be confounded with symptomatic bronchitis from dyspepsia, with emphysema of the lungs.—Diagnosis of the tuberculous infiltration from pneumonia and from pleurisy.—Diagnosis of the more advanced stage of the disease from empyema and from dilatation of the bronchi.

The mean duration of phthisis has been estimated by a celebrated French physician at about two years, for the class of patients admitted into Hospitals. This estimate is probably too favorable. Two-thirds at least of this class of patients die during the first year, and one-third during the first six months of the disease.* In the higher ranks of life, it is probable that the disease is less rapid in its progress, but there exist no data for comparison. I have seen the disease in the highest class of so-

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liver is usually anemic. According to Bowman, the cells of the healthy liver contain a small quantity of fat. (*See Appendix, Pl. I., Fig.* 13, *B.*)

The deposit of fat in the heart, under the pericardium, has been found (by Bizot) to coincide singularly with the fatty degeneration of the liver, and especially in tuberculous phthisis. The two conditions usually coexist, and they are both, and to a most remarkable degree, more common in females than in males. They cannot be connected in any decided way with a general deposit of fat in the system. Frequently the subjects of this condition are in the last stage of general emaciation. Thus, in 35 males, the heart was much loaded with fat only 4 times. In 42 females this condition existed 23 times. Twenty-nine of these 42 females were much emaciated, yet in 14 the heart was loaded with fat; but in 13 women, in whom a considerable general tendency to fat existed, 9 had the heart much loaded with fat. In 25 women with tuberculous phthisis, 11 had the heart loaded with fat; but in 11 males who had the same disease, not one had a fatty heart.

* Of 307 cases,

99 (one-third) died during the first six months.
100 (one-third) died between six and twelve months.
67 (between one-fourth and one-fifth) died from the end of the first to the end of the second year.
Thus two-thirds died during the first year. *Louis.*

Of 215 patients,

22 (one-ninth) died during the first six months.
66 (one-third) died between six and twelve months.
Thus nearly one-half died during the first year.
London Hospital for Consumption

ciety terminate fatally in less than three weeks from the first development of any symptom. I have known a case which was under the care of a medical friend during the long period of thirty-five years before it terminated fatally from a recent and abundant deposit of tubercles.

When you are called to a case at the onset of the disease, you can hardly judge of its probable duration. The general condition of the patient, the rapidity of the emaciation, the state of the pulse, the signs of a more or less extensive deposit, the knowledge of the progress of the disease in other members of the family: these and other circumstances may give you an idea of the probable progress of the case, and which, if you are wise, you will keep to yourselves. When the disease has existed for a long time, you may judge something of the future by the past, and indulge the hope that past checks in the progress of the case will continue to recur, that favorable influences once felt may continue to be enjoyed. You can judge, at all events, whether you have a chronic case to deal with, one in which the vital powers stand up stoutly against the ravages of the local disease. But even in this case, do not attempt to give a positive opinion as to the duration of life. Many cases that appear favorable become suddenly very unfavorable cases. Finally, even in advanced cases do not prophesy rashly. Many cases which seem very near their termination, unexpectedly improve. I have learned by experience never to calculate upon the future in this disease. Indeed, look at some of the unfavorable circumstances which may occur to a patient whose condition promised nothing immediately alarming—a pleurisy with copious effusion, a pneumothorax, a chronic peritonitis, intestinal ulceration, severe hæmoptysis, and, in children, hydrocephalus—any of which, in a few days, may change entirely your ideas of the probable duration of a case, and be even followed by a rapidly fatal issue. Indeed, there are good and bad influences exerted upon this disease, which are often unexpected, and often unknown, except by their effects.

Another important question presents itself. Is phthisis a curable disease? and if so, what is the proportion of cases which recover? The general impression in the medical profession is,

that a patient with phthisis is doomed to death. If those cases only are considered in which the disease is strongly marked, and which is so advanced in its progress that the diagnosis is easy, this opinion is, on the whole, well founded; yet even under these circumstances unexpected recoveries take place. I shall never entirely despair of the life of a patient with phthisis, when I recollect what I once witnessed in this Hospital. A patient was admitted with phthisis. The disease was perfectly well characterized, and in its most advanced stage; a large and well-marked abscess existed under the right clavicle. Indeed, the signs of this lesion were so distinct, that I was in the habit of calling the attention of the students in attendance to them as perfect in their characters. On one occasion, as I approached the bed for this purpose, I found the patient, who had been gradually sinking, in such a state of exhaustion, that it seemed to me improper to disturb him. He was bolstered up in bed, with his head falling upon his shoulder, breathing with great difficulty, bathed in perspiration, and with a rapid and feeble pulse. He looked like a dying man. The next day, my attendance ceased, and after two months was again commenced. On entering the ward, the house physician called my attention to a man, dressed, walking about the ward, apparently stout and well, although somewhat pale. He asked me if I recognized the man. I did not. To my great astonishment, I found that it was the very case of phthisis I had left two months before, apparently dying. This astonishing improvement had not, however, as yet resulted in perfect recovery. There was still some cough, the pulse was somewhat accelerated, and the countenance not entirely healthy. The physical signs still existed under the right clavicle, but were much less marked than before. The patient congratulated himself on his restoration; but I by no means intend to present the case to you in this light. His improvement was astonishing, but he was not entirely cured. If nature was not interrupted in her efforts, he probably regained a tolerable degree of health, so that he may have returned to his usual pursuits. But perhaps a new deposit of tubercles may have taken place in the lungs which did not terminate so successfully.

I have known a number of patients, during the last fifteen years, who have had the evidences of phthisis, and sometimes in an advanced stage, who finally recovered, and are now in the enjoyment of good health. Not perfect health, for such patients carry with them for a long time, perhaps for life, slight traces of their original disease, too slight often to attract the attention of those who, ignorant of their previous history, do not watch them carefully. They do not generally regain quite their former vigor; they are apt to cough a little at times, and perhaps experience a little dyspnœa. But they are fully able to attend to the ordinary business of life.

What light does post-mortem examination throw upon this subject? For the past fifteen years, I have been in the habit of examining the lungs of all my patients dying of every form of disease, independent of phthisis, for the traces of phthisis that has been cured. I have been astonished at the number of cases which have presented evidences of this favorable result. I can say that it is not uncommon to find in patients who have died of various diseases, and in which no suspicion of tuberculous disease existed at the time of death, cretaceous masses, few in number, at the summit of the lungs, and sometimes, but more rarely, cicatrices in the same situation, and commonly existing with these cretaceous masses. Sometimes no tubercles proper can be discovered, sometimes a few are noticed, and sometimes they are more abundant, and evidently of recent origin. Chronic disease of every kind probably favors this deposit, and it is therefore not surprising that patients who have, years ago, suffered from this deposit, and who have recovered from it, except so far as the ancient traces of the disease exist, should, near the close of life, experience a new deposit, which is, for the most part, latent. I have recently been much gratified by the perusal of an essay by a young French physician attached to the Salpétrière Hospital of Paris, the Almshouse for aged women. This physician found in one hundred women, all above sixty years of age, and dying of various diseases, fifty-one who presented the curative indications of tuberculous disease, and chiefly by the formation of chalky concretions. This result did not surprise me, as it would have done many of the profession, who believe

that tubercles are equivalent to a death-warrant. Indeed, I am inclined to think that as the diagnosis of tubercles has become more certain and satisfactory, that many cases will be found presenting undoubted evidences of phthisis, which yet recover, and that the common expression, "The patient could not have had phthisis, because he recovered," will cease to be believed.

I do not wish to state this case too strongly to you. I admit, as all must do, that phthisis is a most fatal disease, and that the prognosis is always unfavorable. I admit that a patient who has experienced one attack is liable to another. Yet facts, and especially anatomical facts, prove beyond a doubt that cases do recover, and that these cases are not very rare. Take as cheerful a view of this melancholy disease as circumstances will permit. I remember a gentleman, now alive and well, many of whose brothers and sisters had died of phthisis, who was attacked with the symptoms of the disease in the most decided manner. These symptoms continued for about two years, when he expectorated a chalky concretion. His improvement coincided so distinctly with this occurrence, that he would hardly believe that the little mass was not a bit of cracker which he thought he must have accidentally got into the air-passages, and that the irritation of this substance had caused all his symptoms. I have known other similar cases. You must not suppose, however, that all the cretaceous matter is expectorated in these cases. A small mass has perhaps formed in an abscess, or, situated near a bronchus, has caused ulceration of this tube, and thus escaped by expectoration. It is simply an indication of what is going on in the lungs.

The chief difficulty in the diagnosis of phthisis is in the early stage, before the rational and constitutional symptoms have become strongly marked, and when perhaps the physical signs are entirely wanting. This absence of physical signs is principally owing to the dissemination of the miliary tubercles through the lungs. A case has occurred to me during the past year which impressed this fact most strongly upon my mind. I examined the lungs after death, and found them studded throughout with an immense number of small tubercles, and yet during life no physical signs of their existence could be discovered. The chest

resounded well on percussion, the respiratory sound seemed unaltered, except that a degree of dulness existed at the base of the right lung, with slight bronchial respiration and a feeble crepitation. These signs, however, were not those of tubercles, but those of pneumonia; and, in fact, this portion of the lung presented the appearances of a lobular pneumonia after death. In this case the deposit existed in both lungs, and extended equally to every portion of them, so that no advantage could be derived from a comparative exploration of the two sides of the chest. But the same thing happens sometimes when the scattered deposit is more limited in extent and in degree. In these cases you must depend upon the rational and the constitutional symptoms, and these may still be insufficient to satisfy your doubts. The cough may have assumed the character of that of bronchitis from the beginning, having been attended by a mucous expectoration, the dyspnœa may be slight and pain in the chest absent. So with the constitutional symptoms. Emaciation, pallor, loss of strength, may be far from being decided symptoms, and the pulse, if accelerated, may be only slightly so, or only so during the evening. Still, it seldom happens that a case is involved in such obscurity. Almost always something exists which will enable you to entertain a very strong suspicion of the true nature of the attack, if not to attain to perfect certainty. Thus, hæmoptysis may have occurred, perhaps, even before the cough commenced. This is a most important diagnostic symptom. I have already spoken of it. I will therefore only repeat now, that nineteen times probably in twenty it is connected with tubercles in the lungs, and that in most of the cases, rare indeed, in which it occurs from other causes, as heart disease, gangrene of the lungs, it is attended by other symptoms quite distinct from those of phthisis. It may, perhaps, also occur in certain cases of cancer or of dilatation of the bronchi, rare forms of disease. In these cases the diagnosis may be more difficult.

The family predisposition of the patient must also be considered. If several of the family have already died of the disease, this fact must have its influence in doubtful cases. A careful study of the pulse will often aid you in the diagnosis. I have

seldom found it entirely free from excitement, even at the outset of the disease. In those cases in which it seems to be calm and natural in frequency, I have reason to believe that there has been, naturally, a slow pulse. There are persons whose pulse is, naturally, not more than sixty in a minute. In them, a pulse of eighty in a minute is an accelerated pulse. When, therefore, you find the pulse rather slow in a patient whose case is suspicious, you should ascertain, if possible, what the usual rate of the pulse has been during health. When the pulse is natural during the early part of the day, and before the patient has been excited by exercise, or by other causes, you may find it quickened in the evening, or from slight exciting causes. It is true, this may be owing to mere nervousness, especially in young and delicate females, and particularly at a first examination. This fact must not be overlooked. On the other hand, it is very common to find the pulse permanently accelerated, and not only so, but with a sharp irritable stroke. Besides, you may discover that in the morning the patient is pale and languid, with occasional flushings during the day, and especially during the evening.

But this obscure and doubtful condition will not continue. In a great majority of cases, you will find more or less dyspnœa and pain in the chest, emaciation distinctly marked, a pale and unhealthy countenance, a tendency to night-sweats, and the gradual development of physical signs at the summit of the lung.

I have occasionally met with cases in which the rational and the constitutional symptoms were well marked, and had existed for a considerable time, yet without presenting any physical signs. I have known these cases recover rapidly, especially when placed in favorable circumstances. You might suppose, in these cases, that no tubercles existed; but this is not a probable estimate of the case. It is more reasonable to suppose that a few tubercles were deposited in the lung, and that their transformation into cretaceous masses had occurred; a result by no means uncommon, as I have already attempted to prove to you. The patient may, indeed, have expectorated some of these little masses, and his improved health has coincided with this fact.

It may be said, with truth, that the rational and the constitutional symptoms, when clearly developed, are of much more value in the diagnosis of phthisis than the physical signs, although, if these signs are present, they add very much to our clear understanding of the case; they often change doubts into certainty.

There are certain cases of phthisis in which considerable infiltration of tuberculous matter into the pulmonary tissues takes place, and which very often attacks the lower and posterior portion of the lung, besides being attended with acute symptoms from the beginning. You will perceive at once the resemblance these cases bear to pneumonia. Indeed, the diagnosis is sometimes quite difficult. You find that your patient has been seized with febrile symptoms, that he has rapidly lost strength and flesh, and you find, in addition, dulness on percussion and bronchial respiration at the base of one lung, while the superior portions of the chest disclose no evidences of disease. You find the cough urgent, and more or less pain in the chest. Indeed, you might readily say, here is a case of pneumonia. But look at the case a little more closely. What is the hereditary tendency? Has the patient had hæmoptysis? Is there any appearance of rusty sputa in the expectoration? Is there any crepitus at the base of the affected lung? Is there a tendency to night-sweats, to irregular chills, with a marked remission of the febrile symptoms in the morning? You can readily, I hope, attach a proper value to the answers which may be given to these questions. If the patient has had hæmoptysis; if his febrile symptoms assume the hectic, rather than the continued form; if, in the progress of the case—and this is usually rapid—a mucous rattle, and, finally, the evidences of a cavity are present in the lung; and especially if the signs of tubercles develop themselves at the summit of either lung, you cannot doubt that phthisis exists.

These cases, which present so much the aspect of a subacute pneumonia, and which are sometimes so rapid in their progress that in a month an abscess may exist in the lung, are not always so rapidly fatal as you might suppose. The patient sometimes rallies, gets better, and may live for a time. I have known a

patient live a year, and perhaps longer, with the lung in this condition.

The dry cough which sometimes exists might lead you to suspect a pleuritic effusion in these cases, especially if the respiratory sound should be feeble rather than bronchial, as may happen. But this will distinguish the case from pleurisy—the dulness does not extend beyond a vertical line drawn from the axilla. In pleurisy it occupies, more or less completely, the lateral portion of the chest. Besides, you will not detect egophony.

There are cases of bronchial irritation which may lead to the suspicion of phthisis, especially when connected with dyspepsia. A dry cough, some dyspnœa, wandering pains in the chest, and, perhaps, a tendency to lassitude, and even slight emaciation exist; yet without febrile excitement, without acceleration of the pulse, unless excited by nervousness, and with no physical signs whatever of tubercles in the lungs. A characteristic feature in these cases is despondency, so different from the recklessness or indifference so generally noticed in phthisis. So that whenever you find a patient disposed to make much of his condition, and fearful of the result, you should always suspect the nature of the case. I do not mean to say that phthisical patients are not sometimes desponding. They are sometimes so if they are dyspeptic, an affection which seems to throw a gloom over every disease which it complicates. In these cases, you may find that symptoms of gastric derangement are evident enough, and that they have preceded the pulmonary symptoms. But it is not unusual, when the functions of the lungs are prominently affected, that the gastric symptoms become quite indistinct, so as to be easily overlooked. The class of cases to which I have now alluded should be carefully discriminated. They are by no means rare, and they are readily cured, or, at least, are unattended by danger.

A slight degree of emphysema at the summit of the lungs, if attended by an irritable condition of the bronchi, and by some degree of dyspnœa, as it commonly is, has been sometimes mistaken for phthisis; and in this case, it is the physical signs that are apt to mislead. You find under one of the clavicles a certain degree

of dulness on percussion as compared with the opposite side of the chest. You hesitate as to the meaning of this. But listen to the respiration where this apparent dulness exists. It is pure, distinct, natural. While on the opposite side, where the percussion is so clear, the respiratory sound is feeble, indistinct, and, very likely, more or less masked by a sibilant rhonchus. The fact is, the dulness which you thought existed is the natural resonance; while the clearness on percussion in the opposite side is an unnatural resonance from emphysema. I remember the case of a young lady, who was brought to me by her parents, supposing that she might be suffering from incipient phthisis. The symptoms, both rational and constitutional, were so different from those of phthisis that I did not think it worth while to examine the chest, a duty, by the way, which I neglected, and the contrary to what I would recommend to you. Her friends had brought her to me to have her chest examined, and they were not satisfied with the opinion I gave them until this was done. On percussing under the clavicles, I was struck with a degree of dulness under one of them, and I was at first somewhat startled by it. But the case was one of those which I have just been describing; a case of slight emphysema, limited to the upper portion of one lung—the dulness was apparent, but not real.

I have seen cases of pleurisy which, after their recovery, have left behind them a trifling, dry cough, some dyspnœa, a tendency to pain in the chest after fatigue or exposure, which have excited some apprehension that tubercles might exist in the lungs. But if you find the pulse calm, if no febrile symptoms are present, and the patient has not emaciated; if you find only the usual physical signs of a cured pleurisy—a degree of dulness on percussion, a feebleness of the respiratory murmur at the base of the lung, and, perhaps, some contraction of the side of the chest in which the pleurisy has existed, you need feel no apprehension. The symptoms will gradually subside, especially if the patient can enjoy the advantage of a favorable climate.

Cases of pleurisy, I am disposed to think, are sometimes followed by the symptoms of tuberculous disease. I have already spoken to you of this fact, and contrasted pleurisy with pneu-

monia in this particular. It may happen, that tubercles are already existing in the lung before the pleurisy is developed. If the pleurisy is double, this is still more likely to be the fact. But at all events, in many cases, there are no evidences of tubercles until after the development of a latent or subacute pleurisy, which, instead of being readily cured, as you might hope from its mild character, passes, insensibly, into a tuberculous affection, marked by progressive emaciation, hectic, increased frequency of the pulse, indeed, by all the symptoms of phthisis. The pleuritic effusion, in these cases, tends to obscure the physical signs of phthisis, producing dulness on percussion and a feeble respiration, particularly at the lower portion of the chest. But this respiration becomes bronchial, a mucous rattle develops itself in time, and very likely the upper portions of one or of both lungs present the ordinary physical signs of phthisis.

When the other acute affections of the lungs complicate incipient phthisis, they sometimes possess a certain diagnostic value from their mode of development. This is true, I think, particularly of pneumonia. You will remember that pneumonia, during the early adult period of life—the period of tubercles—seldom attacks the summit of the lung. It is the base, or the central portion, that is usually attacked. When, then, at this period of life, say from the age of twenty to thirty years, pneumonia attacks the summit of the lung, always think of tubercles. Inquire into the previous history of the patient. Has he had cough or hæmoptysis? Notice the expectoration, whether it contains more blood than is usual in pneumonia; observe if the tendency to night-sweats and to a morning remission is more distinct than is usual in a purely inflammatory disease; and when the pneumonia has disappeared, which it usually will do readily enough, mark if any physical signs are left behind—any dulness, however slight, any alteration in the respiratory sound, any increased vocal resonance. You will be able to judge from these symptoms, and from the subsequent condition of the physical signs—for pneumonia leaves no physical signs after it, whether your primary suspicion, derived from the seat of the pneumonia, is confirmed.

And so it is with bronchitis. The mucous rattle, which, when it exists, is characteristic of the disease if seated at the base of both lungs, and unattended by dulness on percussion, or change in the respiratory sound, may be heard only in one lung—in that in which tubercles exist, and perhaps only at the summit of the lung. A mucous rattle in one lung only, and in a chronic case of pulmonary disease, points very distinctly to the existence of tubercles in that lung. It is still more significant if it exists only at the summit of the lung, or if existing all over the lung, it is more marked and abundant at the summit than at the inferior portions of the lung. Again: when the mucous rattle exists in both lungs in phthisis, it is still at the summit that it exists most distinctly, and thus becomes an evidence that tubercles are deposited in both lungs.

It is the development of a mucous rattle at the summit of the lung which marks more particularly the period of tuberculous softening. When this process commences, the bronchi in the neighborhood of the deposit are inflamed, a mucous secretion is established, which becomes more and more abundant, until it may pass into a gurgling rattle. It is this local bronchitis which, indicated by a mucous rattle at the summit of the chest, is one of the best established evidences of phthisis, in a somewhat advanced stage of its progress. At an earlier period the physical signs may have been indistinct; indeed, the indications from percussion and from the respiratory murmur may still be obscure, when the development of a mucous rattle, slight at first, heard perhaps only at intervals, will remove all doubts, and establish the physical diagnosis on a true basis. Generally, it is true, that when the disease is so far advanced, other physical signs of tubercles in the lungs are pretty evident.

I have seen a false diagnosis made in certain cases of pneumonia at the summit of the lung in which resolution was going on. There is a degree of dulness on percussion, a trace of bronchial respiration, and a well-marked mucous rattle under the clavicle, and in the supra-spinal region behind. Exactly, in fact, the physical signs you will sometimes meet with in an advanced case of phthisis. It is by trusting too much to the

physical signs, and not looking attentively into the previous history of the case, that such mistakes are made. The history in such cases reveals an acute attack, which is not the character of the symptoms in phthisis, unless in very exceptional cases. I remember, many years ago, when I was a Dispensary physician, that I was called in, accidentally, to see a child, whom I hastily examined, and finding a limited dulness on percussion, a bronchial respiration, and a mucous rattle under the clavicle, I pronounced the case to be phthisis in an advanced stage. Some time afterwards, when in the same house, I saw the child apparently well, and found on examination that every physical sign had disappeared. This, then, was a case of pneumonia; a rare case as to its location in a child, and which became rapidly and completely cured.

Chronic pleurisy, with pus in the cavity of the pleura, is sometimes mistaken for phthisis. Indeed, when a pleurisy commences, as it sometimes does, without much pain, or other prominent symptoms of acute disease, and with a dry cough; when hectic symptoms develop themselves, and finally, expectoration ensues of opake and purulent mucus, the patient all the time emaciating and losing strength, the symptoms bear a very close resemblance to those of phthisis, especially to cases in which hæmoptysis has not occurred. It is not to be wondered at that the old physicians, who did not practise auscultation, or attend to the physical diagnosis of thoracic disease, should frequently make mistakes in these cases. But with the advantages of physical diagnosis, this error could hardly be committed, certainly not in a simple case. But tubercles and pleurisy may exist together, and then the peculiar physical signs of each affection must be sought for. I am not afraid of repetition, when I can aid you, by so doing, in establishing the principles of a correct diagnosis. A patient has tubercles in his lungs—latent, perhaps; a double pleurisy, with effusion, occurs; this should excite a suspicion that tubercles are connected with the mischief. A single pleurisy occurs in a delicate person predisposed to tubercles. It is obstinate, it does not yield to remedies, or to time. By-and-by a mucous rattle exists at the upper portion of the chest. Is this owing to the softening of tubercles

in the lung? Very often this is the case, but not always. Let me remind you of what I have already alluded to while speaking of chronic pleurisy. When a lung has been long in contact with a purulent effusion in the pleura, it is liable to a limited inflammation, often attacking the summit of the organ, and which may pass into abscess. The progress of this abscess is indicated by the development of a mucous rattle, and finally by a gurgling rattle with a cavernous respiration. This physical condition once led me to mistake some of these cases. I supposed them to be tuberculous abscesses, knowing how rare it was for abscesses to form in the lung from simple inflammation, and being ignorant that pleuritic effusion not unfrequently led to this result. When the abscess is situated at the summit of the lung, it is, perhaps, impossible to be sure of its real character. It will be doubtful whether it is tuberculous or not. But let me caution you against making up your minds that it is necessarily tuberculous, or you will sometimes find yourselves mistaken.

This tendency to form abscess in the lung, as the consequence of empyema, is an effort of nature to remove the pus from the chest, and sometimes this is accomplished. The abscess opens into the pleura, pus escapes freely and suddenly by expectoration, and a pneumothorax may be established. The cases on record in which the sudden disappearance of the pleuritic secretion coincided with a sudden and abundant purulent expectoration, and which were once attributed to an absorption of the pus, and its subsequent discharge by purulent secretion, a metastatic action now known to be impossible, were probably cases of this kind.

Chronic bronchitis, a rare disease except in old people, can hardly be confounded with tuberculous phthisis. There is no emaciation, no acceleration of the pulse, no hæmoptysis, and no physical signs, unless, indeed, you may find rather a dry respiratory murmur, and perhaps a sibilant rhonchus over the chest, and occasionally, when an acute attack supervenes, a little fever, increased oppression, and a mucous rattle at the base of both lungs. Many persons, especially those advanced in life, expectorate every morning a little mucus—opake, purulent mucus, and yet they live on without suffering more than a temporary inconvenience. I have seen but very few cases of chronic bronchitis

severe enough to make the patient demand medical aid, unless when the case was supposed to be something more serious. But when chronic bronchitis is complicated with dilatation of the bronchi, the case may be quite different, and may simulate phthisis so closely as to deceive the most experienced practitioner. This accident is a structural change—it is an organic lesion; it alters the condition of the lung by compressing the pulmonary tissue, hence hæmoptysis may possibly ensue. The mucous membrane of the bronchi is inflamed in a high degree, hence abundant muco-purulent expectoration, hectic, emaciation, and loss of strength may follow, and the case, indeed, assume very much the rational and the constitutional symptoms of advanced phthisis.

But more than this may happen. The physical signs, instead of diminishing the difficulties of the diagnosis, as they do in so many cases of pulmonary disease, may lead you still more completely into error. Dilatation of the bronchi sometimes produces a certain degree of dulness on percussion, as well as a bronchial, or even a cavernous respiratory sound—with a mucous or a gurgling rattle. If these physical changes exist on both sides, or about the central or lower portions of the lungs only, the question of tubercles might be a doubtful one in your minds. But if this organic change exists only at the summit of the lung, if you find there the physical signs of a cavity, accompanied by most of the rational and constitutional signs of phthisis, you would call the case phthisis, and not hesitate about it either. But you might be mistaken. Yet on the whole, these cases of dilated bronchi are so rare in comparison with tubercles, and the cases in which the physical signs of their existence are developed at the summit of the lung only, are so exceedingly uncommon, that I can only say the thing may happen. I will relate to you a case abstracted from the work of Louis on phthisis, in which this distinguished observer was deceived, as indeed happened in another case of dilatation of the bronchi which I saw in his wards, but of which I have preserved no notes.

A man fifty-nine years old, of rather delicate constitution, and who had been subject to dyspnœa from childhood, had had a cough for more than ten years, especially during the winter. He was pale, with anasarcous legs, feeble, and emacia-

ted. His pulse was accelerated, and he had night-sweats and diarrhœa; he expectorated an opake, greenish matter. The chest resounded well on percussion in every part. But at the summit of each lung the respiration was bronchial, and accompanied by a coarse, mucous, or gurgling rattle, and by increased vocal resonance. The other portion of the chest presented no physical signs of disease, except a sonorous rhonchus. After death, the bronchi, at the summit of both lungs, were found dilated and filled with purulent mucus. Not a trace of tuberculous deposit existed.

LECTURE XIX.

TUBERCLES IN THE LUNGS.

Treatment of tuberculous phthisis.—General indications of treatment.—Influence of exercise; diet.—Climate.—Circumstances which render a residence in a tropical climate desirable or disadvantageous.

In order to form correct notions of the treatment of phthisis, let me recall to your minds some of the leading facts of what may be called the natural history of the disease. You have seen that the primary, the essential fact, is the deposit of what is called tuberculous matter in the lungs—and that the first influence of this deposit is a deteriorating influence. Many persons feel this influence before there is any evidence of local disease. They grow pale, they become emaciated, they lose strength without apparent cause. Or if, as often happens, the local evidences of the disease first appear—the symptoms of pulmonary irritation and obstruction, these, however slight they may be, are equally accompanied by the same indications of impaired health. You will remember, that after a time, a new source of irritation as well as of deterioration is added in the inflammatory condition of the lung which attends the softening and the evacuation of the tuberculous deposit. The pulse be-

first, the breathing of an unirritating atmosphere, protection from cold and moisture, and from sudden atmospheric changes; the careful avoidance of all active exercise of the lungs, or of the muscles of the chest; and of the inhalation of irritating substances; the prevention of gastric irritation; and, finally, the use of remedies which are counter-irritant or sedative in their character.

Let me now call your attention more particularly to the most important of these means of fulfilling the indications of treatment.

The best mode of exercise for phthisical patients is riding on horseback, if the patient be strong enough to enjoy it. But great care should be taken that it is not carried too far. It may be made gentle or violent, just as the patient chooses, and most individuals are apt to tax their strength beyond what prudence would dictate. An easy, pacing movement for a short distance, may be all that can be tried with safety. If too much is attempted, fatigue and excitement follow, and injury, rather than benefit, is the result. Some patients retain vigor enough to make long journeys on horseback, and they are often singularly benefited, if they act with prudence in pursuing their course quietly, and without fatigue or exposure. Other invalids are most benefited by riding in a carriage, and some are able to gain sufficient exercise by walking, care being taken to avoid oppressing the chest by climbing hills or by walking too fast. Persons of susceptible constitution should always be careful to keep in motion when they have once commenced taking their customary exercise. Many more attacks of bronchitis and of pulmonary congestion are induced by a sudden stop when excited by exercise, than by a steady exposure to even bad weather without stopping from some accidental and usually easily avoided cause. So a small draught of air from a door or a window, when the invalid is sitting still, is much more likely to do harm than a walk out of doors, even during the unpleasant season. While patients with phthisis, therefore, should be enjoined to take daily exercise in the open air whenever the weather will permit it, they should receive at the same time, careful directions as to the proper management of this powerful

means of invigorating the constitution. They must exercise with moderation and with prudence. So injurious is the effect of confinement, that I always advise patients in whom the disease is quite advanced, and even during the unfavorable season in this climate, to go out into the open air as much as possible, taking every precaution to guard themselves from exposure, and I have often had reason to feel satisfied with this recommendation, which to some physicians might seem hazardous. But the truth is, an invalid shut up in the house is liable to suffer from numerous and unavoidable, although trifling exposures, which another more accustomed to the open air would hardly feel.

Continued mental and physical exhaustion operate most powerfully in deteriorating the vital powers, and thus exert a very injurious effect on phthisical patients. The active pursuits of business should all, if possible, be resigned, and the invalid should make the improvement of his health his sole business. An attack of tubercles in the lungs is no trifling matter, and when the diagnosis is clearly established, the invalid can hardly make too many sacrifices to regain, if possible, his health. Let him then free himself from the restraints, the anxieties, the exposure of business, and by cultivating a cheerful spirit, and enjoying moderate and well-regulated exercise, or rest and quiet, as the circumstances of his case may require, think only of his health and comfort.

A question now presents itself. Is it better to inform a patient of his true situation, to let him know the worst at once? It is difficult to give a general rule which shall apply to every case. Some persons are very susceptible, and look upon being called consumptive as a certain doom. The effect of knowing the whole truth on such minds is certainly injurious. There is a black cloud before them wherever they go. Others, again, possessed of more firmness of mind, more hopeful, or more indifferent, as the case may be, will receive the intelligence without being disturbed by it. As a general rule, I think that it is best to let the patient know enough of the disease, to regard it as something not to be trifled with or neglected, and that every attention and every sacrifice that can be made to health, should be perseveringly made. But it is better not to

talk about tubercles and ulcers in the lungs, for these are well known in the community in which we live, and their meaning well understood.

But what shall you say to the friends of the patient? Tell them the whole truth without reserve; but be first sure that your diagnosis is correct. A great responsibility rests upon you. If you make a trifling matter of what is really serious, because you are uncertain of the true nature of the case, and therefore are afraid to give an unfavorable opinion, your error will probably soon be discovered, and you will be blamed for it. Or, if you make a serious matter of what is perfectly curable, some quack will perhaps supersede you, and gain great eclat from your incorrect diagnosis. My opinion is, that the friends of the patient should always be treated with perfect confidence. If you are doubtful about the case, say so; ask for a consultation, or wait until time shall have disclosed new facts. If you are certain as to the diagnosis, be equally unhesitating in giving a favorable or an unfavorable opinion of the case. Time, which confirms and strengthens what is true and candid, will always operate in your favor.

The digestive functions of the phthisical invalid must always engage your most careful attention. The diet of the patient, the state of the appetite, and of the secretions, are points of primary importance. The majority of patients suffering from phthisis in its early stage, do not suffer from any marked derangement of the digestive organs, but as the disease progresses the powers of the stomach become impaired, and the deranged condition of the intestinal canal becomes a serious complication. Other patients are dyspeptic before the attack, and continue so. This is a most unfortunate circumstance.

The diet of the patient should be simple, but nutritious. The indication clearly is, to allow him all the nourishment that his digestion will permit him to enjoy. Roasted, boiled, and broiled meats, farinaceous articles of diet, milk, if it is easily digested, which is often not the case, are usually the best adapted to the condition of the patient. There is a popular opinion in the profession, that incipient phthisis should be treated by a rather low diet; that animal food should be abstained from. This, I think,

is a great error. Cases, indeed, occur in which pulmonary congestion, and perhaps inflammation, exist, and in which a rather low diet, for a time at least, may be proper. Indeed, still more active means of reducing congestion may be indicated, as bloodletting; but these are exceptional cases, and the advantage of this treatment is but temporary, and the sooner the necessity for it ceases, the better for the patient. The leading indication should never be lost sight of for a moment. You must sustain the patient; and although occasionally circumstances may modify this principle, it must never be forgotten.

When the disease is more advanced and the digestive powers fail, great caution is necessary in regulating the diet. This is the usual course of things: The appetite first becomes somewhat impaired and capricious; vomiting occasionally relieves it from undigested food, until, at length, a low inflammation of the mucous membrane ensues, which adds greatly to the discomfort of the patient, by causing frequent nausea and vomiting, pain in the epigastrium, and other symptoms less regular in their development. This condition acts unfavorably upon the lungs, by increasing the irritation there. It acts upon them indirectly, and perhaps still more unfavorably, by interfering with nutrition. Thus, when your patient cannot digest his food well, he usually declines rapidly, he suffers more from cough and from expectoration, and, above all, from discomfort of mind, which is the true attendant on dyspepsia. I, therefore, always look carefully at the state of the digestive organs in phthisical patients. When the digestion is good, I endeavor to keep it so by a perfectly simple and digestible diet. As the digestive powers fail, I endeavor to limit the diet still more strictly. Generally, a weak stomach will digest simple, nutritious food better than what are called slops. But I would recommend to a patient to use arrowroot or gruel, if this was the only kind of diet the stomach could digest, rather than more substantial food, which would occasion vomiting and pain.

The use of stimulants in this disease, of wine, brandy, porter, are often useful, but require much caution in their administration. In the early stage, they are seldom advisable. If the digestion is good, if the patient can exercise freely in the open

air, he will be sustained better by nutritious food and by pure air than by artificial stimulants. But in the advanced stage of the disease, when hectic has supervened, and the vital powers are more exhausted, alcoholic stimulants are often highly beneficial. Where the digestion is feeble, brandy usually agrees best. Sometimes porter is preferable, unless there is a tendency to diarrhœa, which porter is apt to aggravate, just as brandy is apt to increase a tendency to constipation. In these latter cases, old Jamaica rum is a good substitute for brandy. Patients are often injured by stimulants, by taking too much, so as to produce excitement, or by continuing them too steadily, when the digestive organs are deranged. Thus it constantly happens in every stage of phthisis, that the tongue will become coated, the appetite impaired, the bowels irregular, from temporary causes. This condition should be at once attended to. Low diet for a few days, rest, and, perhaps, slight alterative and purgative doses of medicine, will be required. Thus the derangement disappears, and every thing goes on as before the temporary attack. It is by watching the effect of diet, by keeping the digestive system in as healthy a condition as possible, that the phthisical patient derives most benefit from medical attendance. If left to himself, he will often persevere in a mode of living, well enough in itself, but requiring modifications and even a complete change, with a change of symptoms.

The same views apply to tonics generally that apply to alcoholic stimulants. They are poor substitutes for nutritious food and for the open air, but they are sometimes obviously beneficial, especially where the digestion is simply feeble. It is difficult to say what form of tonic is to be preferred in these cases, for it is very much a matter of individual experience. Sometimes the vegetable tonics agree the best, and especially the cold infusion of the wild cherry bark. At other times, the mineral tonics are preferred, and when they are tolerated well by the stomach, I think that they are preferable to the vegetable tonics; especially the preparations of iron. There is always more or less anemia in phthisis, which the use of iron tends to counteract. It is often highly beneficial, especially in chronic phthisis. I do not know that one preparation of iron has an advan-

tage over the rest. Those that I would more particularly recommend are the iodide of iron, the Griffith's mixture, and the iron prepared by hydrogen.

A patient, then, in the early stage of phthisis, whose previous state of health has been good, and whose constitution is naturally vigorous, needs no stimulant or tonic, when his digestion is good, and he can exercise freely in the open air. But if he be naturally delicate and feeble, these remedies may be useful, even at an early stage of the disease, and especially if he has been reduced by severe hæmoptysis or by acute disease which has passed away. In the advanced stage of the disease, these remedies are more frequently required, when they, certainly, if used with discretion, add much to the life and comfort of the sufferer. They are useful when the stomach is simply weak and not inflamed, when they are not followed by excitement, and when the secretions of the alimentary canal are in a healthy condition.

The influence of a residence in certain climates or regions of country on the development and progress of phthisis deserves also your most careful attention. The first advantage that a change of climate presents is the ability to breathe a less irritating atmosphere, and less exposed to sudden vicissitudes of temperature. But this is not the only advantage. The endemic diseases of the countries under consideration are equally important in their influence upon the disease. The extensive experience we have already gained on this subject proves, conclusively, that regions situated in the same parallels of latitude, and exposed, apparently, to the same meteorological influences, present very different results in their influence on phthisis, and that this difference is owing, in part, at least, to the prevalence or not, of malarious diseases. It also seems probable that a long-continued exposure to a tropical climate by a northern constitution, exerts a far less favorable influence on the tuberculous diathesis than was once supposed. Indeed, it seems to be established, that when phthisis is well developed, a continued residence in a tropical climate exerts a decidedly unfavorable influence. Formerly, it was supposed that if a person predisposed to phthisis, or with the disease actually developed, could reside,

permanently, in the West Indies, for instance, that his chances of life would be much increased. This has been proved to be a mistake. The British soldier, who leaves home, apparently in good health, for the West Indies, dies much more frequently with phthisis than if he had remained at home. The American soldier, quartered at our southwestern posts, or along the southern shore of the Atlantic, dies more surely with phthisis, than if exposed to the chilly and changeable climate of our northern and northeastern frontier. No one would believe this, did not facts prove it beyond all question. The cause of this difference seems, by both the British and American reports, to be the prevalence of malarious diseases, which, by deteriorating the constitution, open the way to the deposit and to the development of tubercles in the lungs. Still, this general influence is not the only point to consider, for there are regions where malarious diseases are common, and yet phthisis is not prevalent, as in the East Indies, which present a striking contrast to the West Indies in this particular. It may be, that in the former countries, the malarious influence is more immediately fatal to life, so that constitutions broken down by the gradual influence of this poison are more rare. One very curious fact is established; that liver disease is much more common in the East Indies. Can this act as a preservative against phthisis? I have already mentioned to you the singular fact, that confirmed drunkards, who almost always have liver disease, seldom have tubercles in the lungs; while phthisical patients seldom have disease of the liver, except the fatty degeneration, which may be regarded as an excess of a normal condition of the organ. Are, then, chronic diseases of the liver and of the lungs in any degree antagonistic in their influence? The considerations that I have presented to you seem to render it possible that they may be so.

If then a patient, with a strong predisposition to phthisis, or with the disease in the incipient stage, goes to reside permanently in a tropical climate, what will be the probable result? If he goes into a malarious region, at least in this hemisphere, he will probably die the sooner for the change. If he goes to the Mediterranean, as, for instance, to Malta, to the Ionian Islands, the

same fate will probably await him. If, on the contrary, he goes to the East Indies, and particularly to Ceylon, or to the eastern shore of the Bay of Bengal, his chances of life, so far as phthisis is concerned, will probably be much increased. But will he escape the prevalent tropical diseases of this climate? Will not fever, or dysentery, or disease of the liver, carry him suddenly out of existence? This is certainly a very important consideration. Is it not avoiding the chances of immediate danger, for a danger more remote, but equally great?

For my part, I cannot see that permanent residence in a tropical climate affords much chance of permanent benefit to those predisposed to phthisis, or who are suffering from its early stage. It certainly does not to those in the advanced stage of the disease.

But, if a permanent residence in a tropical climate is not to be recommended to the phthisical patient, can he derive no advantage from a temporary residence? Will he not, by avoiding the damp and chilling influence of our winter and spring months, by a residence in a tropical climate at a season when malarious disease is not prevalent, and when the heat is not oppressive, escape in a degree both dangers, and thus materially improve the chances of life? There can be no doubt of this. If proper cases are selected, and proper management is adopted, great good will be experienced—life will be prolonged, and some cases, I believe, permanently cured.

What, then, are the conditions most favorable to the patient? If the disease is in its early stage, if it is limited to one lung, and to a small portion of that lung, if the disease is not hereditary, if no local complications exist, and if the progress of the case has been slow and with occasional interruptions, if the patient has still considerable vigor of constitution, the case may be looked upon as a favorable one. If, on the other hand, the disease has commenced at a favorable season of the year, and has advanced steadily and rapidly notwithstanding this circumstance, if both lungs are affected, or if the tuberculous deposit is very extensive in one lung, if cavities exist, and the usual train of secondary complications have ensued, then the case is unfavorable.

The earlier, then, the period of the disease,* the slower its progress, and the more simple and limited it is, the better are the chances of improvement from a change of climate.

But there is this to be considered. A patient may have the advanced stage of the disease, he may have a well-formed cavity at the summit of one lung, while the other portions of the lungs present no evidences of disease. There is reason to suppose that a single and a limited deposit has taken place, which has gone on to the formation of abscess, without any new deposit in its train. This is, certainly, a favorable case. Nature has already done much. These are the cases in which you will find cicatrices in the lung. Let a patient in this condition be encouraged to try a change of climate for the winter, that nature may be less liable to be interrupted in her favorable undertaking. The patient who goes from home with an abscess, may return home with his lung cicatrized.

Again, you have a patient whose disease is equally limited, and who has expectorated chalky concretions, and with this has shown evidences of improvement in his condition. Let him go away, also, for the winter. The danger is, in all these cases, that a new and perhaps extensive crop of tubercles will be deposited; and if the patient is exposed to the deteriorating influences of bad weather, and of confinement to the house, this

* The following table, constructed by Dr. Renton, a physician of Madeira, presents some interesting facts in relation to the effect of a residence in that climate on phthisical patients:

Patients with tuberculous lungs	56
Died at the Island	30
Left the Island	22
Still there	4
Threatened with pulmonary disease	108
Remained free from symptoms	93
Fell off	13
Lost sight of	2

This table probably does not present the facts precisely as it should do. Many of the patients probably had tubercles in the lungs in the early stage. Other patients very likely were affected with bronchitis only, or with some other affection independent of tubercles. The cases described as tuberculous were probably most of them advanced cases of phthisis, in which the disease was not only advanced, but extensive. We could judge much better of the question if we knew how well Dr. Renton was acquainted with physical diagnosis.

accident will occur, and thus destroy at once all hope of recovery.

Thus, a limited deposit of tubercles, although in an advanced stage, and in which nature has already made an effort to cure, presents a favorable case for the trial of a change of climate.

It is very important for you to have a clear idea of the classes of patients most likely to be benefited by this change. For a residence far from home, even for a season, is a matter which involves so much expense, so much inconvenience, that it should never be recommended, or encouraged, unless there is a fair prospect of benefit. The fate of many patients who go abroad is sad indeed. I have known them to die even during the outward passage; or if they arrive in safety at their destination, they linger on, getting gradually worse, and die in a foreign land. If you make a proper discrimination in your cases, this accident will seldom happen. I do not know of more than one patient of mine who has died from home, during a period of fifteen years. I believe this good fortune has been owing to a careful adherence to the rules I have pointed out to you.

I may mention another circumstance which may be regarded as authorizing a favorable opinion of a change of climate. If the disease has shown a decided tendency to pause in its progress, indicating an effort on the part of nature to resist it—this may be regarded as a favorable indication. I will again also call your attention to the healthy condition of the digestive organs, to the mildness of the constitutional symptoms, and to the absence of complications in other organs, as adding very much to the prospective advantages of a change of climate.

The usual advice I give my patients is this. Pass the winter months in the interior of Florida; Jacksonville is, perhaps, the best place that now offers accommodations to pulmonary invalids. When the heat of the weather becomes oppressive, as is usually the case in March, move a little northward, but still keep clear of the sea-coast. Spend a month or two at Aiken, in South Carolina. Travel slowly northward, and reach Richmond, Virginia, about the beginning of May. Do not return to the Northern States before the 10th of June.

Florida is, undoubtedly, the best climate in our own country

for pulmonary invalids during the winter months. I prefer it to the West Indies, because it is nearer home, easy of access and return, and because it is our own country. To those who prefer the West Indies, Santa Cruz is, perhaps, the best place. The society is chiefly English, and the accommodations better than those usually found in these islands. Many pass the winter at Havana; but the northerly winds which sometimes suddenly prevail there are very objectionable. I suspect, if suitable accommodations could be found on the south side of the island of Cuba, that it would prove an excellent location for pulmonary invalids, but none such exist there. The same advice should be given to invalids going to the West Indies as to those going to Florida, not to remain there after the heat becomes oppressive, but to cross over to the main-land, and pass some time in the southern localities I have mentioned.

It is of the greatest importance that patients should not return to our northern climate earlier than the tenth of June, a period which my own observation has fixed as the real commencement of our summer; and the caution is the more necessary, because patients are always anxious to return as the warm season approaches. A long absence from family and friends, the enjoyment of improved health, is a great temptation to their prudence. But the good effects of a whole winter's residence is sometimes lost by a premature exposure to the chilly and variable weather, and to the easterly winds of our April and May. Disease, which had become quiescent, is excited again to make new progress, which, perhaps, never experiences another check.

Taking a view of all the cases that are decidedly benefited by a change of climate, but few, comparatively, are so far restored to health by the experience of a single winter passed in the South, that they can safely remain at home the next winter. Many are obliged to return winter after winter, and finally get rid of the disease, or keep it so much in check that it makes but little progress. I suspect that most of those who are permanently and rapidly cured, are not cases of true tuberculous disease of the lungs. You may ask, is this experience worth much? I answer, it gives the patient the best chance of ultimate recovery, or it prolongs life even when it does not effect a perfect cure.

I shall not enter into a consideration of other more remote regions, because I have had but little experience of their comparative efficacy. Louis recommends Pau, at the foot of the French Pyrenees. I have known patients derive great advantage from a residence there. Another of my Parisian friends, Dr. Barth, a very judicious physician, entertains a high opinion of Hyères, near Toulon. Nice, and Italy generally, have been gradually losing the reputation they once possessed as favorable residences for pulmonary invalids. They possess many attractions, but the climate is too changeable.

The island of Madeira is undoubtedly one of the best residences with which we are acquainted, especially as a permanent residence for pulmonary invalids. The sea-coast possesses a tropical winter climate; the higher land in the interior of the island, accessible after a few hours' ride, affords a delightful summer residence.

I have known a few patients who have passed the winter on the southwestern coast of England, at Torquay, and who experienced great benefit from it. There is this great advantage possessed in an eminent degree by Torquay—and the same is true to a certain extent of the south of France—all the domestic comforts of life are to be found there: good accommodations, a fine country, and all the advantages of a high degree of civilization are worth considering.

If now we turn our attention to the colder regions of the globe, we shall find that the influence of cold upon the production of phthisis is much more limited than might be expected. In the northern regions of Europe, the disease seems to be comparatively infrequent. What the influence of such a climate as Norway and Sweden may be upon those predisposed to phthisis, or who are suffering from the early stage of the disease, and who have removed there from what are called more temperate latitudes, remains yet to be determined. The English government is now in the habit of sending phthisical patients from their military posts in the West Indies to Canada, and, as it is said, with advantage. But the proof of this is as yet wanting. We are beginning, however, not to dread the influence of a dry, cold climate upon this disease. One of the most successful

cases of incipient phthisis which has ever fallen under my observation occurred in a gentleman, a merchant of this city, who passed the winter in the northern part of the State of Pennsylvania, engaged in dragging logs of wood from the forest through the snow. A gentleman who had a decided attack of phthisis left this country, and returned to Stockholm, his native city, and there, as I have been informed, his health rapidly improved. I have found also by experience that the dry, cold weather of our early winter often agrees very well with this class of patients, and I have learned from it not to recommend too early a departure to the South. This, it is true, does not apply to all cases. Some patients feel the influence of cold to be unfavorable, particularly those who have not been much accustomed to exercise in the open air. Such cases had better leave the North in October, or early in November; but others may safely stay in this region until December, or even until January. A great advantage is gained by this course; a long winter residence from home is materially abridged, and as the spring months are much more injurious than the early winter months, it is better to prolong the period of return, rather than to hasten the time of departure. Many persons, indeed, do not experience the unfavorable influences of our climate until March; to them a comparatively short residence from home, and in a comparatively northern region, as, for instance, at Aiken, South Carolina, and afterwards at Richmond, Virginia, will be the most appropriate course. To every one, however, the advice should be given not to return to the North before the 10th of June.

Facts seem also to prove, that a sea life, in tropical regions, is eminently preservative against the development of phthisis, as well as highly beneficial to those who are suffering from an attack of the disease. If our naval surgeons, imitating the example of the late Dr. Forry of the army, would give us a statistical report of the comparative prevalence of phthisis in the navy, a very important and, as I believe, cheering task would be accomplished. Dr. Turner, an English naval surgeon, has attempted this for certain stations of the British navy; and taking the West India station alone as a guide for comparison, it will appear that the proportion of sailors attacked with phthisis, as

compared with the troops in garrison, is less than one-half, and the proportion of deaths not more than one-third. I have already, when speaking of the causes of phthisis, alluded to certain circumstances—the short period of enlistment, and the facility of being sent home as invalids—which will, no doubt, increase the apparent difference in favor of sailors. But making all due allowance for these circumstances, the old opinion of the favorable influence of the sea on phthisis seems to be confirmed by statistical inquiries on a large scale. It has often occurred to me, that should a vessel, of suitable size and accommodation, be chartered to cruise during the winter and the spring in tropical latitudes, that the advantages of such a trial would be very great to the pulmonary invalid.

LECTURE XX.

TUBERCLES IN THE LUNGS.

Treatment of phthisis.—Means of checking local irritation.—Value of particular remedies in the treatment of phthisis.

The results of statistical inquiry seem to prove, that while the development of phthisis is intimately connected with malarious influences, the acute affections of the chest—bronchitis, pneumonia—exert comparatively little influence in its production. While speaking of the causes of phthisis, this remarkable and unexpected fact has been distinctly brought forward for your consideration. But you must not be led to suppose that the disease, when fully developed, receives no impulse from these causes. Every day's experience proves, that bronchitis especially, exerts an unfavorable influence upon the progress of phthisis. Many patients whose disease is progressing slowly, or is, perhaps, stationary, find all their symptoms aggravated by getting a cold, as it is called. Hectic, loss of appetite, diminished strength, increased emaciation, are the result of this accident, and if the attack is severe, it is a long time before the patient

regains his former position; indeed, perhaps, he never regains it. These attacks are, commonly, attacks of bronchitis, attended by more or less pulmonary congestion. Sometimes pneumonia exists, and, what is much more seriously to be feared, pleurisy, with effusion. It is during the spring that such affections are most common in our climate, but they are not uncommon even in tropical climates. Many pulmonary invalids suffer from them during a winter residence in southern regions.

It is for this reason, that patients suffering from phthisis should be particularly careful to guard against exposure to sudden atmospheric changes, to draughts of air, and the remaining at rest in the open air when heated by exercise. There is a steady, deteriorating influence in a cold, damp climate, and there is the sudden, but often permanent effect of accidental exposure. No climate can protect the invalid from the bad effects of the latter influence, unless he exercises great and constant caution. Without prudence, the chances of improvement in a southern region are hardly better than at the north.

Other causes connected with the atmosphere exert an unfavorable influence upon the lungs of phthisical patients. The inhalation of irritating gases, of turpentine, of mercurial vapors; exposure to the dust of animal substances and of mineral substances, and the inhalation of filamentous vegetable matters, as, for instance, the cotton dust, exert an injurious influence on the lungs of phthisical patients, by irritating the bronchi and leading to pulmonary congestion. The influence of these local irritations on the disease, when it is once established, is sufficiently evident, although their influence as a predisposing cause of the disease is not, as yet, so clearly established.

Active exercise of the muscles of the chest is injurious to the pulmonary invalid, and so is the active exercise of the voice. The latter mode of exercise is beneficial, probably, when only a predisposition to pulmonary disease exists, but it becomes highly injurious often, when tubercles are deposited in these organs. A patient with tuberculous disease of the lungs, will often be more exhausted by a long effort to talk, than after protracted general exercise of the body.

When speaking to you of the kind of diet best suited to the

class of patients now under consideration, I endeavored to impress upon you the importance of guarding against gastric irritation, and of checking any tendency to diarrhœa, however slight they may be. Patients with phthisis are very liable to attacks of gastric derangement, which always act unfavorably upon the lungs. Diet will of course do much to check this tendency, but occasionally medicine is required—sedatives, sometimes leeches to the epigastrium, if inflammation exists, and sometimes mild mercurials, if the secretions are much deranged. The diarrhœa of phthisis sometimes comes on and progresses very insidiously. It begins with a slight degree of looseness in the stools, without increased frequency, but gradually they become more frequent, as well as more loose, and the medical attendant is surprised to find himself called to treat a diarrhœa of considerable standing, and often of great obstinacy, which had escaped his observation because it had not been spoken of by the patient.

I wish now, having called your attention to the general indications of treatment, and to the means of fulfilling these indications, to consider the effect of certain remedies which are employed in the treatment of this disease.

In the first place, I willingly admit that no remedy has as yet been discovered which appears to exert any specific influence upon tubercles, either in preventing their development, or in promoting their cure. Antimony, digitalis, iodine, have all had their day of imaginary success, and all been forgotten. Cod liver oil, the present popular remedy, is destined to experience the same fate. It has not, in my opinion, any specific influence in phthisis. It has not, in my experience, performed any wonderful cures. I do not, however, mean to deny its usefulness in this disease. It certainly sometimes appears to diminish the emaciation, to improve the appetite. It is good nourishment, nothing more, and I think it very probable that other kinds of oil, equally well prepared, may exert the same beneficial influence.

Those practitioners who regard the deposit of tubercles as one of the effects of inflammatory action, and who mistake the symptoms of irritation which they create for those of inflammation, have been led to adopt a practice in the early stage of this dis-

ease highly injurious to the welfare of the patient. Venesection has been resorted to, and repeated from time to time, and leeches have been applied to the chest, and low diet recommended, with antimonials, with the vain hope of removing the imaginary cause of the disease. Digitalis is another antiphlogistic remedy which has been much abused in the treatment of incipient phthisis. The effect of this agent in reducing the frequency of the pulse for a time, has led to its use in phthisis, as if an accelerated pulse was anything more than a mere symptom of an important organic change in the lungs—as if the temporary influence of a poison could make amends for the debility and the gastric disturbance it is sure to induce.

Cases of active pulmonary congestion, attended by hemorrhage, sometimes occur, especially at an early period of the disease, and in those whose previous health has been good, which requires active depletion. The attack borders very closely upon inflammation; the pulse is full and strong, the skin hot, and there is pain and oppression in the chest. In such a case venesection is plainly indicated, with antimonials in nauseating doses, cool drinks, cool air, and perfect rest.

In other cases, leeching and cupping to the chest will agree better with the indications, or may be required after a general bleeding. But however strongly depletion may be indicated in these cases of active congestion, you must never forget to keep in view the probable future progress of the disease, its exhausting tendency, and the necessity of preserving all the constitutional vigor that is possible to resist its encroachments. Daily experience confirms the fact that the effects of depletion are slowly recovered from by phthisical patients. Moderate hemorrhage, even of an active kind, will often cease by the use of the simplest means—rest, low diet, and cool air. Other cases may require the temporary use of tartar-emetic, or of the acetate of lead, and perhaps are benefited by the application of a few leeches to the chest, followed by a small blister. It is fortunate for the patient when these milder remedies are sufficient to remove the symptoms of congestion.

A case came under my notice about two years since, which will impress upon your minds the consequences of the free de-

pletion which is sometimes adopted. A young man of moderately strong constitution was attacked with the usual symptoms of phthisis. For two months the disease did not appear to progress with much activity. He was then attacked with pulmonary hemorrhage. He was bled freely from the arm three times at short intervals: after this, the hemorrhage ceased. He was bled again and again, from fear that the hemorrhage would return! It is probable that the loss of blood from hemorrhage and from twice repeated bleedings had induced the reaction, the throbbing pulse, which attends anemia. He was bled, then, twice for anemia! He was next advised by his physicians to pass the winter in the West Indies. He came as far as this city, from Vermont, where he had been ill, and I was called to see him. I found him as pale as a corpse, only able to sit up for a short time, and with a very feeble and rapid pulse. I did not think he could live more than twenty-four hours. By the use of stimulants he rallied a little, and lived nearly a week.

Many cases of hemorrhage are passive in their character. There is no fever, no unusual excitement of the pulse. Sometimes in these cases, the hemorrhage occurs suddenly, and subsides suddenly, and the patient feels actually stronger and more buoyant in his feelings than before its occurrence. Hemorrhages of this kind, no doubt, often exert a beneficial influence on the lungs. But they may be excessive, or may continue too long. Perfect rest and a cool atmosphere, with a light and simple diet, are all proper in these cases. But in addition to these means, you may often use with advantage the spirits of turpentine in small and repeated doses (ten to twenty drops in mucilage every two hours), or the vegetable astringents, as tannin, or matico, or the acetate of lead.

Some years ago, the practice of treating phthisis by inhalation, especially by iodine combined with conium, was highly recommended by a distinguished English physician, Sir Charles Scudamore. His apparatus for inhalation was procured for the use of this Hospital, and the remedy was administered to a patient, as I am informed, with a cavity in one of his lungs. The patient improved very much, and was discharged. Some months afterwards he returned with pericarditis, of which he died. In

examining the lungs after death, a cicatrix was found in the place of the former abscess. This was regarded as a very favorable result for a first case, and the remedy was afterwards perseveringly tried, but no similar second case ever occurred.

And so it is, I believe, with inhalations of tar and of chlorine gas, old remedies, revived from time to time to fall again into oblivion.

There is a class of remedies, called expectorants, which are much employed in the treatment of phthisis, but which, I think, should be banished almost altogether from the treatment of the disease. Many physicians are in the habit of prescribing to their phthisical patients, as a matter of course, a cough mixture, containing a variety of expectorant remedies, combined with more or less opium. The different quack remedies so much in vogue have, probably, most of them, the same composition. But what influence can these remedies have upon a disease like phthisis? They may quiet the cough for a time—the opium they contain will do this—and thus deceive a hopeful patient with the idea that his condition is really improving. But the long-continued use of these remedies is sure to impair and to disturb the digestive function, without actually benefiting the lungs. Many are the consumptive patients who have bitterly regretted the use of these remedies when they look back on the sad experience of the disease from which they are suffering.

But you must not discard these remedies entirely. An occasional attack of bronchitis, complicating the original disease, may be benefited by their temporary use.

The opium which enters into the composition of these cough mixtures I regard as a most valuable palliative remedy in phthisis. I hardly know what could be done to procure relief in many cases of advanced phthisis without its aid. Many a weary night has been soothed by its influence. The misfortune is, that there is no substitute for it when it happens to disagree with the constitution of the patient, as it sometimes does, producing watchfulness instead of composure; excitement, with itching of the skin and nausea, instead of quiet. Other sedative remedies, such as hyoscyamus, conium, hydrocyanic acid, possess very limited powers in controlling the symptoms of irritation, when

compared with opium. Happily, when one form of the remedy does not agree with the patient, another form may be more successful; and there are many forms in use which may be resorted to in turn.

Much as I value this remedy, I always try to defer its use as long as possible, and then to use it in the smallest possible doses. In the earlier periods of the disease, it is often uncalled for, and even at an advanced period, its constant use is by no means always required. If the patient can enjoy a reasonably quiet night, he is better without the remedy than with it. My habit is to begin with the smallest dose that will procure rest; and it is surprising how small a quantity will often produce the desired effect. A single drop of Magendie's solution of morphine will sometimes quiet a patient who is unaccustomed to the use of opium. Give him an ordinary dose, and it is true that he falls asleep, and very likely does not awake until morning. But he awakes with a feeling of confusion in the head, with a furred tongue, with loss of appetite, and probably with constipation of the bowels. Certainly, these effects follow the regular use of what are called ordinary doses of opium. Neither are these effects likely to be beneficial to a phthisical patient.

Another point of practice which should be attended to in the administration of opium to phthisical patients, is, that it should not be taken at night as a matter of course. The patient may enjoy a good night's rest without resorting to its use. Let him make the experiment, and resort to the remedy when he fails to obtain rest. Thus, by administering small doses of opium when the comfort of the patient requires them, and by gradually increasing the quantity as the symptoms become more urgent, you can promote the temporary comfort of your patient without material injury. In advanced cases of the disease, when all hope has fled of even any temporary improvement, and a condition of distress is the constant and prominent condition of the unfortunate patient, then opiates, frequently repeated, and in large doses, are alone capable of affording relief.

Opium is also the most valuable remedy we possess in controlling the diarrhœa which exists so frequently in connection with phthisis—a symptom which should always be carefully

watched, and checked at its commencement, if possible, by an appropriate diet.

It is a common practice to attempt the relief of pulmonary irritation by blisters, or by the more permanent forms of counter-irritation, as by the tartar-emetic ointment, or by an issue applied to the chest.

I have already stated to you, that the thoracic pain in phthisis was produced by two distinct causes. The most common cause is a neuralgic affection of the intercostal nerves, indicated by flying pains and by external soreness, especially on each side of the spine between the shoulders. This kind of pain is usually relieved by the mustard plaster, or by a liniment composed of spirits of camphor and tincture of aconite, three parts to one part. The more fixed and constant pain in the chest, especially that attended with a febrile exacerbation, is caused by a slight secondary pleurisy, and is most frequently seated in the upper portion of the chest. To relieve this pain, it is generally necessary to apply one or more blisters to the chest.

The application of an issue to the chest, or some other permanent form of counter-irritation, seems to be an appropriate treatment for the relief of internal irritation. The numerous cases, especially cases of diseased articulations, in which this treatment has been evidently attended with great benefit, would naturally lead you to suppose that similar applications would be beneficial in a disease like phthisis. I have been watching the effects of these agents for many years, and have been struck with the great differences in the result. Some patients, in the same stage of the disease, are benefited, others are not. What is the reason of this difference? My own impression is, that those cases in which much secondary inflammation, and especially where a pleurisy with effusion exists, are the cases which are really benefited by this treatment. The pulmonary irritation produced by the tuberculous deposit is not likely to be benefited by it, any more than any other form of irritation. A neuralgic affection of a joint is injured by the application of an issue. It is when chronic inflammation is the chief condition of the disease, that issues are so beneficial; and the same is true of the lungs.

An issue is, however, only applicable to those cases in which the vital powers are still considerable. If the patient, in an advanced stage of the disease, is worn out with hectic and with restlessness, an issue will only increase these symptoms. I have relieved patients, not unfrequently, by healing an issue under these circumstances, and the relief is sometimes very decided.

The tartar-emetic ointment is a powerful, but painful counter-irritant, and, therefore, less convenient than an issue. The application of Croton oil to the chest is a milder form of counter-irritation. An occasional blister is sometimes beneficial.

My own impression is, that in this disease all liniments, or applications containing oil, as well as the numerous plasters that are found upon the chests of phthisical patients, are objectionable, as tending to interrupt the cutaneous exhalation. The best application to the chest is, after all, water; cold water, if reaction is easy; warm water, if the patient is feeble. Sometimes this application may be made more refreshing by the addition of a certain quantity of alcohol. An excellent local wash for the chest is also made by mixing half an ounce of the nitro-muriatic acid with a quart of water. These applications should always be followed by dry friction, until the skin becomes dry and warm. This simple mode of bathing the chest with water, and afterwards applying dry friction, is worth all the liniments that have ever been thought of. It should be resorted to, regularly, every morning, when the patient rises from bed. It is not only very refreshing, but to a certain degree preservative against bronchitis and other inflammatory affections of the chest. But if an acute inflammatory attack should supervene, its use should be suspended for a time.

The chest should, of course, always be carefully protected by clothing. A flannel or netted undershirt, or one made of silk, if the skin will not bear well the application of wool, should always be recommended to the patient.

While speaking to you of local applications as a means of relieving pulmonary irritation, I wish to call your attention to an instrument called Jeffries' Respirator. It is composed of layers of fine wire gauze, to be worn over the mouth when in the open air. This is really a valuable aid to irritable lungs,

and is much less used by patients than it should be, owing, probably, to the appearance of the thing. If applied to the mouth before leaving home, the gauze becomes heated by the breath, and the patient breathing entirely by the mouth, the air which is about to pass into the lungs is warmed as it passes through the wire gauze. In fact, the principle is the same as tying a handkerchief over the mouth and breathing through the nose, an expedient, the value of which is well known to those suffering from irritable lungs. There are many patients who cannot ride out in the damp and cold air of winter and spring without beginning to cough, and without experiencing a stricture in the chest. To such the use of a respirator is particularly valuable.

I may mention another instrument which has been used a good deal in cases of phthisis; or rather a practice, for no instrument is necessary. It is the practice of expanding the lungs by a forced inspiration, and then expiring the air by a narrowed passage. It is a quackish invention, but what are its effects? It is said that it will expand the chest if persevered in. This is very possible. But is this likely to be beneficial in phthisis? If the disease is active, if there is a tendency to hemorrhage, certainly not. If, on the contrary, the disease is quiescent, or there is reason to believe that concretions are forming in the lungs, it may be useful in assisting the healthy action of the lungs. Where the lung has been compressed by a pleuritic effusion, which has, at length, been absorbed, this forcible expansion of the lung is likely to prove highly beneficial in assisting nature to overcome the effects of compression.

A very troublesome symptom, especially in the advanced stage of phthisis, is the tendency to night-sweats. They often occur, disappear, and recur without apparent cause. But sometimes the cause of their appearance is evident. A chill in the early part of the day, an attack of indigestion, fatigue, will induce them. In many cases they are certainly aggravated by too much bed-clothing at night, so that a little care in guarding against this may cause them to cease. Sometimes they are partial, occurring chiefly about the neck. In these cases an astringent local bath, as of alum, will remove them for a time. When

they are more general and more profuse, the mineral acids often relieve them, and the acetate of lead with still greater certainty. Opium, which procures sleep, seems rather to increase the tendency to perspiration, for it is during sleep that it occurs. The patient who lies awake at night, will not be annoyed by perspiration, unless, indeed, at a very advanced period of the disease.

The treatment of phthisis, to return again to the two indications which I have already pointed out to you, should consist in the use of such means as are calculated to sustain the vital powers and to guard against local irritation. In the early stage, let the patient seek the open air, and enjoy such exercise as his symptoms will permit. If active pulmonary congestion, or the symptoms of inflammation, ensue, an antiphlogistic treatment should be promptly resorted to, but in as mild a form as the case will permit. Let your patient adopt a mild but nutritious diet, and preserve carefully the tone and the healthy condition of the digestive organs. Use as little medicine as possible, and only to correct certain symptoms, which may be easily removed. Remember there is no specific remedy in phthisis. If the disease is limited in extent, slow and intermittent in its progress, and without complication, let your patient seek a favorable climate during the winter and the spring. In the advanced stage, the same invigorating means must be employed, and as the powers of digestion fail, they may require the support of tonics and of stimulants. Local irritation will also require direct means to check its progress. Opium, in small doses, is the best means you possess. Diarrhœa must be controlled by its use, aided by mild astringents. The debilitating effects of night-sweats must be checked by the mineral acids, or by the aid of astringents; and finally, the close of life must be solaced by simply attending to the immediate comfort of the patient. Thus, by carefully watching the symptoms, by avoiding the causes of debility and of irritation, by opposing their progress by suitable remedies, the life of your patient will certainly be prolonged, and sometimes his more or less complete recovery will reward the judicious exercise of your medical skill.

LECTURE XXI.

CANCER IN THE LUNGS.

Pathology of the disease.—Varieties of cancer.—Scirrhus, Encephaloid, Colloid cancer.—Duration: Symptoms of cancer of the lung, with illustrative cases.

TUBERCLE is not the only heterologous deposit which you will find in the lungs, although it is by far the most frequent deposit. There is another deposit, called carcinoma, or cancer, to which I wish to call your attention in the present lecture.

Cancer presents three distinct forms, which are not different stages of the same disease, but which differ originally, but only in the proportion of the elements which enter into the composition of every cancerous deposit. These elements are a fibrous tissue, and a viscid fluid contained in cells, and called the cancerous juice. It is the difference in the arrangement and in the proportion of these elements which constitutes the chief difference in the appearance of a cancerous mass when divided by the scalpel. If the fibrous tissue predominates, you will find the mass hard and creaking when divided by the knife. You will notice that its cut surface is intersected by white lines, or by larger masses of a dense, white structure. In the midst of these lines, you will discover a finely granulated substance—the cancerous juice contained in cells, and which may be pressed out by the finger, or scraped off by the scalpel, when it often resembles very much in appearance apple-juice. This form of cancer is known as *scirrhus*.

When the fibrous element is less distinct, and the cancerous juice more abundant, the cancerous mass is much softer in its texture. It is often more distinctly granular, and from its resemblance, in many cases, to the substance of the brain, it has been called *encephaloid*. Finally, the fibrous tissue may be still more deficient, or even entirely absent, and a jelly-like mass, sometimes semi-fluid or even fluid, and collected in cells, often of considerable size, and united with cancer cells, may

exist, constituting what has been called the *colloid*, or gelatinous cancer. (See Appendix.)

You must not, therefore, regard these three varieties of the cancerous deposit as different forms of disease, but as the same disease; and you will notice that their most important difference consists in the different proportions of what may be regarded as the proper element of cancer, the cancerous juice.

But the cancerous deposit will be found to differ in appearance from other causes, which may be called accidental. It differs in color. The cancerous juice is commonly semi-transparent and white, with a yellowish tint. It has been compared to apple-juice. But sometimes it is more yellow, even as yellow as pus, from being mixed with a certain amount of fatty matter. A cancerous mass of moderate consistence and deeply tinged yellow looks very much like a tuberculous mass, and it has been mistaken for it. I think it probable that the distinguished pathologist, Bayle, made this mistake, in two of the four cases which he has published. The same mixture of fatty matter may cause the cancerous deposit, when it has become fluid, to look very much like pus, yet without containing a single globule of that fluid. In other cases, the cancerous juice is of a dead, white color, and presents a milky appearance, giving to the cancerous deposit a dead-white aspect, or perhaps it may retain a slight degree of transparency. Again, you will observe that some of these cancerous masses are very vascular, and that their vessels are easily ruptured. Thus, their tint may be rosy, or distinct spots of ecchymosis may exist in their substance. Finally, the mass may appear dark, or even blackish, from the more or less abundant deposit of black pigment. These tumors, thus darkly colored, have been erroneously supposed to constitute a distinct form of disease, called melanosis. But the black pigment, like fat, is a natural element of the healthy system.

The external appearance also of a cancerous mass varies very much. It has a tendency to become lobulated. This is sometimes a very prominent feature of its external appearance. Sometimes it is encysted; sometimes it is deposited in minute particles, not larger than a hemp-seed; at other times it exists

in large masses, which may attain an enormous magnitude; while, finally, instead of existing as a circumscribed mass, it may be infiltrated into the tissues of an organ without distinct limits.

The consistence of the cancerous deposit varies very much at its commencement. It may be very firm and solid, or you may find it in nearly, and perhaps, when thrown out upon the free surface of an organ, in quite a fluid condition. But when it is deposited in a solid form, it has a tendency to soften. There are, therefore, two but not very well-defined stages of cancer, a stage of comparative firmness and a stage of comparative softening, which latter stage may pass into a fluid state, and may become encysted, or opening upon a free surface, as that of the skin, or of a mucous membrane, may form what is commonly called a cancerous ulcer, although it is only connected with ulceration as a secondary process. It is the softer form of cancer that tends most decidedly to the fluid condition. I have found it changed into a thick opake fluid mass resembling exactly lime prepared for whitewashing. Sometimes it is of a yellow color, and then you might mistake it for pus.

The chemical composition of cancer possesses nothing which is characteristic. The most abundant element in its composition is albumen. It also contains some fatty matter and some fibrine, with inorganic salts—as the subphosphate of lime, the carbonates of lime, soda, and magnesia, the hydrochlorates of soda and of potassa, the tartrate of soda, and the oxide of iron.

The microscopists, however, contend that they have discovered a characteristic element of cancer. They maintain, that there is an elementary cancerous cell, which is usually found in great abundance in the cancerous juice, and most abundantly in the encephaloid variety. This is the form of the disease, also, in which it exists in its most perfect development. They state that this cell differs from every other cell, found either in healthy or in diseased structures; in a word, that it is a characteristic cell. It is not found alone, but mixed with other microscopic objects, as fibres, fusiform bodies, inflammatory globules, yellow fat, cholesterine, and with the black pigment to which I have already alluded. The more or less abundant development of these acci-

dental elements influence, as I have already mentioned, the appearance of the cancerous mass to the naked eye, causing it to appear more or less fibrous, or yellow, or blackish, in different cases. It must be admitted, I think, that the labors of the microscopists have already been attended with a great advantage to pathology. If in time, a cancer cell is admitted by the mass of practical men in the profession* as a characteristic of cancer, the diagnosis of this disease will be much better established than it now is. The best pathologists are well aware that the diagnosis of cancer by its external appearances is not always easy. Fibrous tumors, inflammatory indurations, tumors composed of epithelium, have been mistaken for it. If now, the microscope, by detecting a peculiar kind of cell as an essential element of cancer, and existing in all the forms of the disease, can effect what neither chemistry, nor the ordinary mode of examination by the eye and by the scalpel in the hands of the most experienced observers, can discover—that is, the positive existence of cancer in all cases, then a great advantage has been gained. The cancerous cell is easily exhibited under the microscope. You have only to take a little of the cancerous juice, and dilute it with a drop of water, place it between the proper glasses, and you will readily detect the cells. I shall not attempt to describe these cells, because, as I remarked in speaking of the tubercle cell, no description, however exact, can compare with a single glance at them through the microscope. For the best description and the best plates of both the cancer and tubercle cells I refer you to Lebert's work on Pathological Physiology. (Pl. II.)

Cancer, when deposited in the lungs, seems to have its original seat, most frequently, in the cellular tissue of the organ. It is generally of the encephaloid variety. In twenty cases, in which the post-mortem examinations were noted by different observers, sixteen were described as encephaloid, three as encephaloid and scirrhus united, and one as scirrhus and colloid cancer.

* During a recent visit to Europe I inquired of some of the best pathologists in Paris and London, Louis, Barth, Golding Bird, what they thought of the diagnostic value of the tubercle and cancer cell. They were not disposed to admit the claims of the microscopists. But only one of these distinguished pathologists, Golding Bird, is at all familiar with microscopic examinations.

Sometimes, small white masses are scattered through the lung, only large enough to be distinctly recognized by the naked eye. In other cases, you will find masses of a flattened and circular shape, varying in size from a five cent to a twenty-five cent piece of silver coin, which are deposited especially in the sub-pleural cellular tissue. The centres of these flattened masses are frequently umbilicated, and vessels are seen ramifying over their surface through the pleura, which may appear somewhat thickened and tarnished, or remain perfectly natural. It is in cases in which cancer of the mamma has been the primary lesion, that these sub-pleural masses are, perhaps, most frequently found. You may find, also, large masses of cancerous matter, sometimes encysted, but generally pretty well defined, scattered through the lungs. Sometimes it is infiltrated into the tissues of the lungs. In thirteen of the twenty cases to which I have alluded it was deposited in the substance of the lung. In more than one-half of these cases it was the right lung that was affected, viz., in seven cases. In two cases the left lung only was affected; and in four cases both lungs were the seat of the deposit. These cancerous masses are more or less disseminated through the lung, and in seven of the thirteen cases they had affected the whole lung. It seems to me, also, that the deposit has a slight preference for the apex of the lung—far less, however, than is observed in the tuberculous deposit.

There is another mode in which the cancerous matter is deposited in the chest. It is deposited in the cellular tissue of the anterior, or of the posterior mediastinum, where it forms a tumor, sometimes of great size. I shall describe one of these tumors, which occurred in one of my patients, which filled the whole left side of the chest, and which weighed not less than eleven and one-half pounds. These cancerous tumors compress the lung, which is, sometimes, also at the same time the seat of a limited cancerous deposit; they compress the trachea, the great vessels of the heart, the œsophagus. Five times in twenty cases of cancer of the chest, the tumor originated in the posterior mediastinum, once in the anterior mediastinum, and once it appeared to have been developed equally in both these spaces. In four of these seven cases, the lung was also the seat of the cancerous deposit.

In the early period of the disease, and when seated in the lung, the pulmonary tissue seems to be simply compressed. You will be struck with the remarkably healthy appearance of the lung, even in the neighborhood of considerable cancerous masses. This form of the disease seems to convert the pulmonary tissues into its own substance. The larger bronchi, however, resist the longest this influence, and may be seen running through the mass of their natural size, although, perhaps, somewhat flattened in shape. A lung that has become extensively the seat of this deposit, is actually smaller than the healthy lung, a fact not to be forgotten in the physical diagnosis of the disease. The lung shrinks rather than enlarges by degenerating into a cancerous mass. The cancerous tumors, however, which grow from the mediastinum, sometimes attain to a very great size, even distending the parietes of the chest.

Secondary inflammation is of frequent occurrence in recent, as well as in advanced cases of cancer of the lungs. You will frequently find the pleural sac obliterated, either wholly or in part, and sometimes by firm and well-organized adhesions. At other times you will find lymph with serum, and even pus in the cavity of the pleura. The effusion may also be non-inflammatory in its nature, constituting hydrothorax, but then the serum is usually tinged by blood.

Cancer of the lung, like tubercle, seems to undergo a period of softening, during which it excites a secondary inflammation of the lung, especially bronchitis. It is seldom, however, that the softened masses are expectorated, or even that they become encysted in the lungs. But that it is the tendency of the disease to produce ulceration, at least in certain cases, and in other organs, is quite apparent.

In the twenty cases of cancerous deposit in the lung, or in the mediastinum, the amount of deposit was sufficient to have produced physical signs of disease, ten times over the right lung, six times over the left lung.

It is stated by a high authority, that cancer may, in certain cases, undergo a spontaneous cure. Cases have been known to occur, in which the disease, when seated externally, has been entirely removed by ulceration and by sloughing, and a healthy

cicatrix has formed. This process is quite analogous to that which forms the cicatrices which are sometimes found in the lungs after tuberculous abscesses. But I know of no case in which the same process has taken place in the lungs. At other times, the cancerous deposit undergoes a curative change of a different kind. The fibrous tissue becomes more and more predominant in the mass, and finally it occupies the whole mass, and by contracting, puckers the surrounding tissues so as to look like a cicatrix, although it is not one. Or finally, the fatty matter in the deposit may become more abundant, and usurping the place of the cancer-cells, may gradually change the malignant mass into a benignant tumor.

Cancer of the lung is a rare form of disease.* I have myself had but three cases under my care in which the history of the cases during life, and the post-mortem appearances after death have been carefully recorded. These cases I shall describe to you somewhat in detail. The remaining seventeen cases from which I have already, and shall again derive certain statistical facts, belong to different authors.†

The duration of cancer of the lung is very different in different cases. It may terminate fatally in two months after the first development of symptoms, it may continue for four years. In the twenty cases to which I have referred, this was the minimum and the maximum duration. The mean duration, in fifteen cases, was between thirteen and fourteen months. It is probable, however, that in the early stage of the disease, the cancerous matter, like the tuberculous matter, is not unfrequently latent.

It would appear that there is a slight difference in the relative duration of cancer of the lung, and cancer of the mediasti-

* Mr. Tanchou, in his Statistical Report of Cancer, states that during a period of eleven years, 382,851 persons died in the Department of the Seine—in Paris, and in two neighboring towns. Of these, 9118 died of cancer, and *only seven* of cancer of the lungs.

In the Report of the Inspector of the City of New York for three successive years, I find 245 deaths from cancer, in a total mortality of 45,890 (the cases of cholera in 1849, still-born children, premature births, and casualties having been deducted), but not one from cancer of the lungs.

† Stokes, Hughes, Syms, Bayle, Martin-Solon, Andral, Bennett, Taylor, Carroll.

num. Twelve months being the mean duration of the former, and sixteen months the mean duration of the latter form of the disease.

The mean age in twenty cases was forty-one years, the maximum being seventy-two years, and the minimum twenty-three years.

Five of these patients only were females, which is a result that could hardly have been expected when you remember the much greater frequency of cancerous diseases in females than in males.*

The cases of cancer of the lung may be divided into two classes. The first class may include those cases in which the lung itself is the seat of the deposit, and in which the symptoms are those of pulmonary irritation and oppression, as in most cases of chronic pulmonary disease. The second class may include those cases in which the cancerous deposit takes place in the loose cellular tissue of the mediastinum, constituting a tumor sometimes of great size, and in which the symptoms are rather those of pressure than of irritation. These cases may produce many of the symptoms of aneurism, or of disease of the heart, or of liquid effusion into the pleural sac.

These remarks are, I think, important, as placing before you a natural division of the subject, although I by no means mean to state that the symptoms of these two forms of thoracic cancer are not sometimes more or less confounded with each other. Cancer of the lungs, more than any other important disease of the chest, is wanting in characteristic symptoms and physical signs; and the true diagnosis of the disease is more often founded, not only on the anomalous character of the indications furnished by the physical signs, and by the rational symptoms, but by the existence of tumors discovered in other parts of the body, and by the evidences of local venous congestion.

Cancer, when it attacks the substance of the lung, commences very insidiously. There may be a slight cough, dry or attended by a trifling mucous expectoration, some dyspnœa, and almost

* Tanchou states, that of 9118 individuals who died of cancerous affections, 6957 were females. In New York, 50 males to 188 females died of cancer.

always pain in the chest. In the progress of the case, the cough and expectoration become more decided, the dyspnœa and the pain in the chest become also more frequent and severe. Hæmoptysis also frequently ensues. The constitutional symptoms are usually decided. Emaciation and loss of strength ensue, the pulse sometimes becomes accelerated, the countenance assumes a peculiar straw-color, the superficial veins of the neck, of the chest, or of the abdomen become enlarged. Œdema of the face, or of one, or of both arms ensue, often with œdema of the lower extremities; and the patient dies, worn out by gradual exhaustion of the vital powers, and with a constantly increasing dyspnœa, aided, perhaps, by the development of a low inflammatory condition in the chest, or in the abdomen.

The physical signs that you may expect to find in these cases are, dulness on percussion, according to the seat and the extent of the cancerous deposit, often, however, existing over a whole lung, and of a very decided character; a bronchial respiration, increased vocal resonance, and sometimes a rhonchus, and less frequently a mucous rattle. Besides, you will often find the affected side of the chest to be smaller than the opposite side. It is contracted, either throughout, or in some portion of its extent, and often moves less in respiration than the healthy side. It is probable, that in certain cases, the condensation of the pulmonary tissue may be carried so far, that the bronchi even may be obliterated, and then with great dulness on percussion, you would have an absence of all respiratory sound.

I will relate as briefly as possible a case of this form of the disease. I was called on consultation, by Dr. Crane, of Brooklyn, to a gentleman about sixty years of age. It was in September, 1848. The patient was a man of vigorous constitution, and he had habitually enjoyed good health, until the preceding winter. In February he noticed a cough. In the early part of June he began to experience neuralgic symptoms, commencing in the ring and little fingers of the right hand, and gradually extending up the arm to the shoulder, and to the upper portion of the right side of the chest. About the same time, he began to notice dyspnœa, and his cough continued, with a trifling mucous expectoration. His attendant physician, Dr. Crane, dis-

covered a marked dulness on percussion, limited to the upper and anterior one-third of the right side of the chest. After a time the cough ceased, but the dyspnœa and the neuralgic symptoms increased. No hectic symptoms had ensued, the pulse was usually about 76 in a minute, and there was but little emaciation. When I was called to see the case in September, the patient had been confined to his room only for two or three weeks, and principally on account of the dyspnœa.

I found the patient in bed, and lying on the left side. His countenance was pale and rather sallow, the dyspnœa was considerable, but his chief complaint was of the pain in the right side, increased by lying upon that side. This pain was chiefly felt at the inferior edge of the scapula, and passing round to the mammary region. It also existed at the top of the scapula, and extended down the arm, but was less in degree. This pain was, at times, lancinating, and posteriorly it was described as feeling like a raw surface. It was not immediately increased by percussion, although the next day afterwards it was more decided. There was no sensation of numbness, and no paralysis, although the patient, when he wished to move the affected arm, seized it with the left hand. The pulse was regular, very little accelerated, and the skin was cool. No hectic symptoms existed.

On examining the chest, the space between the right clavicle and the corresponding nipple was flattened, depressed, while the supra-clavicular region appeared rather more full than natural. Over this space, the dulness on percussion was very marked, and the respiratory murmur was harsh, and with a prolonged expiration. Behind, over the same extent of lung, the respiration was bronchial. The vocal resonance, in front, was increased, with a peculiar hoarse vibration. At the summit of the lung, behind, it was less decided; no rhonchus or rattle was noticed. Over the rest of the chest, the percussion and the respiratory sound were natural.

I visited the patient again after an interval of two weeks. He had been rapidly failing. Hectic had supervened. The pulse was 120 in a minute. The respiration was 36 in a minute and labored, and at times the patient experienced paroxysms

of dyspnœa. He lay upon the back, and upon the left side. Lying on the right side still increased his pain. For a few days he had experienced dysphagia, with a feeling of obstruction, without pain. He coughed only once or twice in the twenty-four hours. I made no physical examination of the chest, as motion, and especially percussion, increased the pain, which was still very severe. He died four days after this visit, and about eight months after the first development of chest symptoms. The dysphagia ceased before death.

On post-mortem examination, there was slight œdema of the trunk and of the lower extremities. There was evident contraction of the superior portion of the right side of the chest, and this extended slightly to the lateral region. The right lung was universally attached to the ribs by old adhesions, and these were the strongest at the summit of the lung. Nearly the whole upper lobe of this lung was incrusted by a thick shell of scirrhus, which appeared to have been deposited in the sub-pleural cellular tissue of the lung. This crust was irregular, nodulated, and very firm. It creaked when divided by the scalpel, and contained the cancerous juice. In front, it was half an inch thick, and posteriorly and internally, where it was in contact with the pericardium, it was still more thick and more nodulated. It did not exist posteriorly and externally at the summit of the lung. This deposit penetrated, at one point, by a distinct mass into the substance of the lung. The substance of the upper lobe, generally, was condensed and unaerated, and softened into a grayish pulp in some portions. At the apex, a small cavity, lined by a membrane and containing a grayish detritus, existed. White lines were observed running through this lobe of the lung. Numerous minute white bodies were scattered through the middle and lower lobes of the right lung, and throughout the whole left lung. The pulmonary tissues in which these small masses existed were somewhat congested, but were otherwise healthy. Under the right pleura costalis several distinct umbilicated cancerous masses were noticed. A small portion of the first and of the second ribs, near their connection with the transverse processes of the vertebræ, were carious; also a small lateral portion of the fifth or of the sixth rib. The heart was healthy.

The liver contained five or six small cancerous tumors, the largest about the size of the common white bean. These were seated, for the most part, immediately beneath the peritoneal coat. The remaining abdominal organs presented nothing remarkable. The head was not examined.

Let me call your attention, while the case is fresh in your minds, to some of its more striking peculiarities, which enabled me to diagnosticate it at my first visit. The case could most readily be mistaken for a tuberculous case. And this mistake was the more easy, from the fact that the patient had been supposed to have been tuberculous in early youth, and had spent two years at sea, where his health was restored. The limited dulness on percussion, the bronchial respiration, and increased vocal resonance at the summit of the right lung, with the flattening of the chest—the rest of the lungs being apparently healthy, and—no evident disease existing in any other organ—seemed to point out what I encounter very frequently—a limited tuberculous deposit of the right lung. The slight cough, and the dyspnœa, the deterioration of the vital powers, might readily lead to the same conclusion. But how were the severe neuralgic pains in the chest—and in the affected portion of the chest—to be explained? Not, certainly, by the existence of tuberculous disease. They indicated some tumor in the chest, irritating the intercostal nerves. Was it an aneurism? No signs of an aneurism existed. The cachectic and sallow appearance of the patient evidently pointed to some formidable disease, and a cancerous tumor of the lung was the most probable conclusion to which I could arrive. There was no hæmoptysis in this case. This might have occurred. This symptom is quite as frequent in cancer as in tubercle of the lungs.

Is it also probable that the softening of the cancerous mass in the lung had occurred at the time of my first visit? I think that it had not, at least to any considerable extent. There was no physical sign of softening, and especially no rattle. It is probable that this commenced, and progressed rapidly during the fortnight that intervened between my first and my second visits. What happened during that time? The patient grew rapidly worse, hectic supervened, and the dyspnœa increased. Had I

been permitted to make a physical examination at my second visit a few days before death, what should I, probably, have discovered at the summit of the right lung? A mucous rattle; perhaps a cavernous respiration, with gurgling.

This cancerous mass at the summit of the lung produced some of the symptoms of a compressing tumor. Indeed, did not its irregular and firm crust give it the physical characteristics of a tumor? Thus, notwithstanding the contraction of the chest, there were symptoms of pressure—neuralgia with caries of the ribs, dysphagia.

This form of disease sometimes commences suddenly, with acute inflammatory symptoms, which, yielding imperfectly to treatment, leave behind them symptoms of chronic pulmonary irritation and obstruction, which are caused by the cancerous deposit. Dr. Taylor's case, reported in the London Lancet, is an illustration of this fact, besides being one of the best reported cases on record. I am, therefore, tempted to place before you some of the more important facts of this case.

The patient was a woman forty-one years of age, who was first seized with acute pain in the loins, dependent, as the post-mortem examination proved, upon cancer of the lumbar vertebræ. A month after this, and three months before her admission to the Hospital, she was attacked with inflammation of the right side of the chest, attended with pain, dyspnœa, violent cough, and expectoration of blood in clots. She was treated antiphlogistically, with relief, but *the cough and the expectoration of blood continued*. These symptoms disappeared for a time, but the hæmoptysis returned.

When admitted to the Hospital, she was emaciated, sallow, without fever, but with a small, soft, and frequent pulse. She had neuralgic pains in the back and in the lower extremities, an abdominal tumor and dysphagia. *But no cough or expectoration* existed; *no dyspnœa, no pain in the chest.* On percussion, however, the right side generally was dull. Very dull over the upper part, with a sense of resistance, like that experienced by striking a solid body. A little below the clavicle, however, and at the side, just below the axilla, percussion yielded an amphoric resonance, and below the mamma, the sound had a tympanitic

character. The dulness on percussion did not pass the median line. In all the upper two-thirds of the right side of the chest, before, behind, and laterally, as well as above the clavicle, a pure and strong bronchial respiration existed. In the lower portions, bronchial respiration was also heard, but it was less loud, and seemed to be produced in smaller tubes. The vocal resonance at the upper portion of the lung was very strong, but the tremor felt by the touch was less than natural. Over the left lung the percussion was natural and the respiration puerile. There was no enlargement of the superficial veins, no œdema of the upper portions of the body.

A month after admission to the Hospital, she had cough with expectoration, dyspnœa, and soreness in the right side of the chest, while the bronchial respiration became less distinct. A mucous rattle developed itself over the anterior surface of the affected lung, which increased both in the number and in the size of the bubbles, until, finally, a short time before death, a distinct gurgling was perceived about the middle of the lung anteriorly, and a large muco-crepitant rattle was diffused over the whole anterior surface of this lung. At the same time, the dulness on percussion somewhat diminished, and the sense of resistance on percussion was less. She sank rapidly, and became delirious before death.

On post-mortem examination, partial adhesions of the right lung existed, and about eight ounces of a turbid, thickish, dirty-gray fluid, with lymph, existed in the cavity of the right pleura. The right lung was rather smaller than natural, and did not collapse. Its color was a dirty, dark, greenish-brown: it was hardly crepitant, and exceedingly lacerable. In some portions, it was granular, in other portions, broken down into a dark-colored, dirty-looking pulp. Its odor was offensive. A foreign deposit was copiously diffused through nearly the whole lung, mostly in small masses, of a dirty-gray color, with their surfaces marked by small, vascular arborizations. In the lower lobe, there was a small cavity, lined by a false membrane, and communicating with a bronchus. About the root of the lung there was a considerable mass of encephaloid matter, extending as high as the top of the sternum along the vertebral column. The

right bronchus passed through it, also several of its subdivisions, and one of them was observed to be much diminished in its calibre. The left lung was quite healthy. Cancerous deposits existed in the stomach and in the kidneys; and an encephaloid tumor, the size of an orange, was found over the lumbar vertebræ, and which had partially destroyed one of these bodies.

Here you have a case of cancer of the lung, commencing with acute symptoms, and among them hæmoptysis, and followed by an apparent recovery, except that this last symptom returned. At the time of admission into the Hospital, the cancer of the lung was latent, yet the physical signs clearly indicated that serious and extensive disease existed in the lung. But what was its nature? In the first mentioned case, there was neuralgia of the chest, which was of great assistance in the diagnosis; in this latter case it also existed, but remote from the chest. But an abdominal tumor existed, which must have been of great assistance in the diagnosis. There was also dysphagia. You might inquire what was the cause of the tympanitic sound on percussion, in the lower portion of the chest, at the first examination? It was probably owing to a pneumothorax, although the author of the case entertained a different opinion. The affected lung was probably also more or less in a state of gangrene.

LECTURE XXII.

CANCER IN THE LUNGS.

Cancer in the mediastinum; illustrative cases.—Appreciation of the more important symptoms of cancer of the lungs and of the mediastinum.—Diagnosis.—Treatment.

In the preceding lecture I invited your attention more particularly to the consideration of cancer of the lungs. Let me now call your attention to another form of thoracic cancer, or that which originates in the mediastinum, and which often forms a large tumor, by which not only the lungs, but the other organs

in the chest are compressed. Cancer of the substance of the lung, indeed, produces sometimes the symptoms of pressure. It certainly did so in the first case I have related, in which it induced caries of the ribs and neuralgia, and also obstruction in swallowing. In this mediastinal variety of the disease, the tumor may compress the great vessels about the heart, and induce œdema, and even the signs of valvular disease, or it may grow and fill the whole cavity of the chest, and present the physical signs of an empyema.

Let me illustrate this statement by two cases which have occurred to myself.

A gentleman, forty-five years of age, of rather delicate constitution, had been subject to a trifling cough for many years. About two years before I saw him, he began to complain of slight dyspnœa, and of wandering pains about the chest. But his general health was unaffected. Six months before I saw him, he had an attack of severe pain in the left side of the chest, which continued several days. Since that time the dyspnœa has rather increased, and wandering pains in the loins and about the chest have continued. He attended, however, regularly to his business. A day or two before I saw him, in attempting to pull at a rope, he felt something snap in the anterior portion of the left side of the chest. This was followed by intense pain about the third rib, in front, with sub-cutaneous effusion of air into the cellular tissue in that region.

When I saw him in consultation with Dr. Du Bois, the effused air was nearly absorbed, and the patient complained of but little pain in the chest. There was, however, considerable dyspnœa. The patient was rather pale and feeble, the pulse was only sixty-five in a minute, and no febrile symptoms existed. On examination, the whole left side of the chest was dilated, intercostal spaces as well as ribs, and it was universally dull on percussion. The respiratory murmur was feeble. I formed the opinion that a liquid effusion existed in the left pleural cavity. After a time, I thought that I heard egophony, and I noticed that the heart was pushed over to the right side of the chest.

I saw the patient again, after an interval of about four months. He continued to linger, but attended more or less to his busi-

ness. His pulse was only eighty in a minute: he had no fever, no night-sweats. At that time the physical signs continued unaltered, except that there was rather more clearness on percussion, and a more distinct respiration under the left clavicle. He continued to decline, and I saw him again after an interval of about three months. He was then mostly confined to his bed. He was pale, emaciated, and disposed to lie only on the affected side. His cough was moderate, and accompanied by a trifling mucous expectoration. He had never had hæmoptysis. He complained but little of pain in the chest, but suffered a good deal from dyspnœa, his respiration being forty in a minute, and chiefly diaphragmatic. The sides of the chest, and especially the left side, moved but little. His pulse had gradually become accelerated to one hundred in a minute; it was feeble. There was also a slight disposition to night-sweats.

At this time, I made a careful examination of the chest, and these are the physical signs I noticed. The whole left side was much dilated, especially laterally, and about the third rib in front. The intercostal spaces were also dilated and tense, as if from strong internal pressure. For two or three weeks, the intercostal space between the second and the third ribs had been observed to bulge during expiration. This I did not detect. The sound on percussion, all over the left side of the chest, was very dull, especially laterally, and about and above the nipple. The respiratory murmur could be heard anteriorly under the left clavicle, and posteriorly above the spine of the scapula. Over a considerable space about the root of the lung, a strong bronchial respiration existed. Over the rest of this lung no respiratory sound could be detected, but a creaking, friction sound existed at the base. No rhonchus or rattle existed in any part of the lung. I thought that I could detect an imperfect egophony in the lateral portion of the chest. On the right side, the heart was felt beating far to the right of the sternum, and a slight crepitus was detected in the mammary and in the axillary regions.

The next day the patient was worse, and was evidently sinking. His pulse was 108, and feeble, his skin was cool. He had

several attacks of faintness. The crepitus in the right lung was more distinct, but was unaccompanied by dulness or percussion. The respiration was more oppressed, there was orthopnœa. The patient died the same evening.

At the post-mortem examination, slight œdema of the feet existed, and the body was much emaciated. I removed the skin and the muscles from the anterior part of the chest in the usual way, and found the second intercostal space so dilated, tense, and even bulging, and yielding *so distinct a sense of fluctuation*, that I did not doubt the existence of a large collection of fluid. But there was no fluid. On the contrary, a large irregular tumor filled not only the left cavity of the chest, but also the mediastinal spaces, and at least one-quarter of the right side of the chest. This tumor adhered but slightly to the parietes of the chest. The left pleural sac contained about half a pint of reddish serum, and the pleura itself was opake and thickened. The left lung, much reduced in size, but otherwise healthy, lay upon the upper and the posterior portion of the tumor. Its bronchial tubes were large and numerous, and seemed to constitute the greater portion of the lung. The right lung presented a pneumonia of the middle lobe. The heart was small and flabby, and was pushed far into the right side of the chest by a kidney-shaped process of the tumor, which left an indentation in the walls of the left ventricle.

This tumor grew from the posterior mediastinum. Its weight was eleven and a half pounds. Its vertical diameter was twelve inches, its transverse diameter was eight inches, its circumference two feet. It was composed, in fact, of five tumors, surrounded by a common capsule, the thickened pleura. The two largest, constituting two-thirds of the whole mass, were of a pale, yellowish-white tint, marbled with spots of a deeper yellow, or reddish tint, firm to the touch, creaking under the scalpel, and yielding, when scraped, a little viscid, yellowish, apple-juice fluid. These tumors were scirrhus. Two of the smaller tumors were of a much deeper yellow color, soft and flabby, lobulated, areolar, with large cells, containing a more viscid, yellow fluid, without admixture of fat. These tumors were specimens of the colloid, or jelly-like cancer. The remaining tumor was more

firm and dry than the rest. It was distinctly fibrous, and in portions bony. The jelly-like matter was infiltrated among its fibres.

The abdominal organs were all healthy, although the left lobe of the liver was reduced by compression to a thin, tongue-like process.

In this remarkable case, the physical signs of extensive internal pressure from some cause were most apparent, while the rational and the constitutional symptoms were most insignificant. Ninety-nine times in one hundred, this cause of internal pressure would have been a liquid effusion into the pleural sac, either of serum or of pus. The trifling constitutional and rational symptoms which existed when I first saw the case, would naturally lead to the idea that hydrothorax existed. Afterwards, when hectic had supervened, the existence of an empyema was more probable. But it was neither of these conditions. It was a solid tumor, cancerous in its nature. I may remark, also, that there was no other evidence of cancerous disease in the system, no other tumor, internal or external, no hæmoptysis, which, had they existed, might have materially assisted in the correct diagnosis of the case.

The cancerous deposit in the mediastinum does not always form a distinct, eccentric tumor, like that which I have just described. It is sometimes infiltrated irregularly into the cellular tissue about the great vessels of the heart, compressing them, and inducing symptoms which might readily be mistaken for ordinary heart disease. I have met with such a case.

An equestrian, forty years of age, of good constitution, but intemperate, and exposed to many privations, was admitted into this Hospital. Six months previously, he was seized with dyspnœa, wheezing, slight cough, pain in the precordia, and with febrile symptoms. These symptoms continued about a week, but they would return at intervals after a condition of comparative comfort. He had been unable to lie on his right side since his first attack.

He presented the appearance of a man suffering from severe chronic disease. He was very pale, and much emaciated. His skin was cool, and rather dry; his pulse was 112 in a minute,

and rather feeble: a general dropsy existed. The feet and legs first became œdematous, about three weeks before his admission. The left arm and the left side of the chest were in the same condition, also the right side of the trunk, but in a less degree. Considerable ascites also existed. The liver extended as low as the umbilicus; there was considerable dyspnœa; the tongue was pale and clean; the appetite was good; the bowels were constipated; and the urine deposited an orange-colored sediment.

On examining the chest, the whole left side was found dull on percussion; the respiratory murmur was absent inferiorly and laterally, and feeble superiorly: egophony was detected. *The side was rather contracted than dilated, and the heart was considerably displaced to the right;* a loud rasping sound existed with the impulse of the heart, having its maximum over the base of the organ, passing into a bellows murmur in the course of the aorta, and gradually diminishing towards the apex.

The dyspnœa increased; the action of the heart was at times irregular; the rasping sound was heard very distinctly along the aorta, but over the body of the heart it became extremely feeble and indistinct; the dropsical symptoms became more decided; the morbid sound over the heart continued, but more faintly; delirium ensued; and death occurred two weeks after admission to the Hospital.

On post-mortem examination, the appearance of emaciation was very marked. Both lower extremities, the left arm, and the left side of the trunk were œdematous. The left pleura contained about three quarts of bloody serum, with a few flocculi of lymph; the pleura was much thickened, was opake, and thinly coated by lymph; the left lung pressed upward, occupied about one-third of its cavity, and adhered to the walls of the chest, both anteriorly and posteriorly, with great firmness; a mass of carcinoma, partly scirrhus, partly encephaloid, granular, of a dirty white color, with irregular yellowish specs scattered through it, exhibiting here and there the rudiments of vessels, and yielding, when gently scraped, a viscid, yellowish-white fluid, occupied the anterior mediastinum, from the origin of the great vessels of the heart to the top of the sternum, and involving these

vessels in its mass. It also penetrated into the posterior mediastinum, involving the trachea and the œsophagus. Masses of the same cancerous deposit, of considerable size, existed in the substance of the left lung. Some of the large pulmonary veins were filled with and obliterated by it, and numerous small masses were scattered through the lung. The pulmonary tissues in the immediate neighborhood of the cancerous masses appeared quite healthy; the right lung was healthy, except that in its lower lobe it contained a few masses of pulmonary apoplexy; the bronchi were somewhat inflamed, and the bronchial glands were much enlarged, and some of them were cancerous.

The pericardium contained twelve ounces of bloody serum, and much lymph; the heart itself was healthy; the calibre of the aorta and of the pulmonary artery, and the cavity of the left pleura, were much diminished by the pressure of the cancerous mass around them.

The liver was healthy, although it descended considerably below the false ribs; a few cancerous masses existed in the pancreas and in one of the kidneys; there was a quantity of straw-colored serum in the cavity of the peritoneum; the other abdominal organs were healthy. The head was not examined.

In this case, you will observe that the first attack was an acute attack with chest symptoms and with febrile reaction, and that these symptoms were mitigated, but showed a tendency to return. I have before illustrated this mode in which cancerous disease of the lung first develops itself. The symptoms at the time of the admission of the patient were those of heart disease —general dropsy commencing in the feet, signs of obstruction of the aortic orifice. You may ask, Was not the rasping sound produced by the pericarditis? I think not. Because the quantity of fluid in the pericardium (twelve ounces) was almost too large to permit a friction of the opposing false membranes. And again, the mode of transmission of the sound along the aorta belongs to aortic obstruction, rather than to pericardial friction. Besides, the contracted state of the aorta and of the pulmonary artery, surrounded by a dense, irregular mass in contact with the sternum, presented the best possible condition for a loud rasping sound, such as was first noticed, but which diminished as the ac-

tion of the heart became more feeble, and perhaps as the pericardial effusion increased.

There is one point on which I have laid considerable emphasis in speaking of this case. With the evidences of internal pressure, the affected side was contracted, although the heart was pushed over to the right side of the chest. You would not expect this in hydrothorax, or even in empyema. You would expect to find the side dilated, if altered at all in size. This is one of those anomalous symptoms which characterize cases of cancer of the lungs.

Martin-Solon has published a somewhat analogous case, which was mistaken for an aneurism of the aorta.

A young man, thirty-one years of age, was attacked with pain in the precordia. About two months after the attack, he presented the signs of a tumor compressing the organs in the chest. Marked dulness in the precordial region, both in degree and in extent, with pain on percussion. A bellows murmur existed in the same region. The dulness on percussion increased, and the bellows murmur diminished. The respiratory murmur over the left lung became indistinct, and dysphagia ensued.

A month afterwards, the countenance of the patient was pale and wan; the pulse, feeble and regular; the respiration, rapid and painful. The chest sounded dull on percussion everywhere, except in the right lateral region. The bellows murmur had ceased, and the patient died in the last stage of emaciation, being able neither to breathe nor to swallow.

The anterior mediastinum was occupied by a mass of encephaloid cancer, which weighed nearly three pounds. It was seven or eight inches long, six inches broad, and of equal thickness. It compressed both lungs, and especially the left lung. Its posterior surface was united to the pericardium, and this again to the heart, which was pushed backward towards the spine, and was only about two-thirds its natural size. No cancerous disease was found in any organ.

You must have noticed, from the cases that I have placed before you, that cancer of the lung sometimes begins insidiously and progresses slowly, with symptoms of moderate pulmonary irritation and oppression, until, at length, an acute inflamma-

tory attack invites attention more decidedly to the condition of the chest; that in other cases, the acute attack may be the first indication of thoracic disease; and finally, that the case may present the characters of a chronic disease during its whole progress, until the period of the softening of the cancerous mass ensues. Then the symptoms become more acute, and the progress of the case is more rapid. You will notice, also, that the constitutional symptoms are often very trifling in the early stage of the disease. The pulse is often unaccelerated—there is no hectic—no very marked emaciation until acute symptoms of an inflammatory action are developed, or until the cancerous mass begins to soften. Then emaciation and loss of strength make rapid progress. The countenance becomes pale and often straw-colored, the pulse accelerated, and hectic may supervene. In certain cases dropsical symptoms supervene, not merely the œdema of the feet, which is common in the last stage of many chronic diseases, but especially œdema of the face, of the upper extremities, and of the walls of the thorax. This condition was noted in five of the twenty cases I have attempted to analyze. In other cases, and not unfrequently in connection with this œdema of the upper portions of the body, you will notice an enlargement of the superficial veins, the jugulars, the veins of the thorax, and even the external epigastric veins. These conditions point to internal pressure on the great vessels of the heart. And you might suppose that they would be chiefly found in the cases of mediastinal tumors. But they appear to me to be equally common in the cases in which the cancerous mass is deposited in the lung itself.

There is another fact that you must not overlook. In many cases, eleven times in twenty cases, there was, during the progress of the case, some internal or external development of a cancerous tumor in some other part of the body, which, if detected, must influence very much the diagnosis in doubtful cases. Solitary cancer of the lungs, even including in this expression cancer of the mediastinum and of the bronchial glands, did not exist in quite one-half the cases.

A straw-color of the skin, and especially of the face, has been regarded, to a certain extent, as characteristic of the cancerous

diathesis. What is its real value as a diagnostic symptom? It is mentioned only six times in the twenty cases now before you. For myself, I consider it as simply an indication of anemia. It exists, equally well marked, in other diseases, and especially in those individuals whose constitutions have been broken down by malarious disease. If it is a condition of anemia, we should expect to find it especially in those cases of cancer of the lungs in which considerable hæmoptysis had existed. And this appears to be the fact, at least to a certain extent. Three of the six cases in which the straw-color existed, had lost a considerable quantity of blood, while none of those who did not present this color had any hæmoptysis. The same straw-color of the face is noticed in other diseases, not cancerous in their nature, of which free hemorrhage is a symptom, as in certain cases of fibrous tumors of the uterus. The complexion, indeed, as the disease advances, usually becomes very pale, and the countenance bears the characteristic stamp of severe organic disease. Sometimes, indeed, the countenance assumes a livid hue.

If now you turn to the rational symptoms of cancer of the lungs, you will find the four symptoms I have already mentioned to you when speaking of another chronic disease of these organs, tuberculous phthisis. These symptoms are pain in the chest, dyspnœa, cough, and expectoration.

In eighteen of the twenty cases now before you, pain in the chest existed. In ten of these cases it was severe. In only three cases, however, was it of a lancinating character. It must, therefore, be regarded as a very prominent symptom in this disease.

In thirteen of the twenty cases, dyspnœa attracted attention, and in twelve of these cases it was a prominent symptom. It is often one of the earliest symptoms, and is usually much increased by the supervention of an acute, inflammatory attack, probably connected with pneumonia, and especially with pleurisy, and by the occurrence of softening of the cancerous mass.

Cough existed in seventeen of the twenty cases. It may at first be insignificant and dry, but as the disease advances it becomes more violent, and is attended by a mucous expectoration. Or it may commence in a decided manner, with an acute inflam-

matory attack, and subside, or even disappear for a time, to return again during the course of the disease. I am not aware that the softened cancerous matter has as yet been discovered in the expectoration. Indeed, we should hardly expect this, when we remember that cavities in the lungs, formed by the softening of the cancerous deposit, is of very rare occurrence.

Hæmoptysis is a very common symptom in these cases. It occurred eight times in twenty cases, and in four of these cases it was considerable in amount. In this respect the disease resembles tuberculous disease of the lungs, and if cancer of the lung was not a very rare form of disease, the diagnostic value of this symptom in phthisis would be materially diminished. In seven of the eight cases in which this symptom occurred, the disease was in the lung itself. In one, the deposit was in the mediastinum, and compressed the lung.

Cancer of the lung is sometimes a more or less latent disease. Cancerous masses are deposited in the sub-pleural cellular tissue, especially when cancer of the mamma has existed, and liquid effusion takes place into the pleural sac, which cannot be distinguished from any other form of liquid effusion, by the physical signs. You would expect to find dyspnœa a prominent symptom in such cases, and, probably, more or less cough would exist. In other cases, the disease is disposed to be latent, from the development of the disease in other organs. There may be no thoracic symptoms, and yet well-marked physical signs of severe thoracic disease be detected, especially dulness on percussion, and bronchial respiration.

The diseases with which cancer of the lung is most apt to be confounded, are tuberculous phthisis, empyema, and aneurism of the thoracic aorta. In phthisis, the disease often commences with nearly the same symptoms, and progresses in a similar manner; and even the physical signs, as in one of the cases that occurred to myself, may be quite analogous. Yet in most cases of cancer, there are some anomalous symptoms which are not met with in phthisis. Thus, in the case to which I have alluded, there was severe neuralgic pain extending down the arm, which at once made me doubt the existence of phthisis. The pulse was also quite natural in frequency, which could certainly not

be expected in a case of phthisis so far advanced, and there was no hectic. The absence of hectic symptoms and a tranquil pulse seem to be rather striking characteristics of cancer before the period of softening, a condition which, probably, does not often occur until a late period of the disease. A leading fact in the history of tuberculous disease is the great uniformity of its seat at the summit of the lung, and its tendency to pass to the formation of cavities. Neither of these facts are true in cancer. Again, tuberculous disease in its progress most frequently affects both lungs so as to be recognized by the physical signs. This is not the case with cancer. The physical signs are seldom detected in more than one lung. Again, the symptoms induced by pressure upon the veins are not observed in phthisis, as they, not unfrequently, are in cancer of the lung. Œdema of the upper regions of the body, enlargement of the superficial veins, are not likely to occur in phthisis. Cancerous tumors, when developed in other organs so as to be felt externally, are usually larger than those of a tuberculous nature. In empyema, the affected side of the chest is usually dilated; there is always extensive dulness on percussion, and absence of respiratory murmur, unless at the apex and at the root of the lung. In cancer of the mediastinum, precisely the same physical signs may exist, and in both diseases you may notice an œdema of the upper portions of the body and enlargement of the superficial veins. In the remarkable case of mediastinal tumor that I have stated to you, the only physical sign that might have led to the suspicion that the disease was not a collection of fluid in the chest was, that the dulness, although universal, was yet most marked under the clavicle of the affected side. This would not readily happen in empyema; the sub-clavicular portion would, probably, sound clearer on percussion than other portions of the affected side.

In cancer of the substance of the lung, the affected side is usually contracted and very dull, and a bronchial respiration is heard over it. This character of the percussion and of the auscultation is sometimes met with in empyema, but it is rare. You may remember a case to which I alluded when speaking of empyema, in which a bronchial respiration existed all over the

affected lung, with universal dulness on percussion, and which was found, after death, to be empyema with ancient adhesion of the lung to the diaphragm. But in this case the side was dilated, which would not have happened, probably, in cancer of the lung; although in empyema the side is not necessarily dilated. I have seen it contracted, even with a considerable effusion of pus in the chest.

Cancer of the lung may be confounded with aneurism of the aorta, or with valvular disease of the heart. If a cancerous mass, too small to produce any external tumor, is infiltrated around the origin of the great vessels of the heart and compresses them, the physical conditions of a permanent organic bellows murmur is produced. How could you distinguish this case from valvular disease of the heart? Not by the sign I have mentioned. But, perhaps, other circumstances might exist. The heart is not enlarged, perhaps, as it would probably be in valvular disease. So in the attempt to diagnosticate a tumor caused by a cancerous mass from an aneurism of the thoracic aorta. How could you do this? Both diseases are tumors, compressing, irritating the organs in the chest. But an aneurism of the aorta has certain favorite seats in the chest, in front about the right third rib, at the top of the sternum, above the clavicle. These are not the favorite seats of cancerous tumors. Again, a bellows murmur remote from the heart, a thrill, and when it presents externally, an expansive pulsation, belong to aneurism, rather than to a cancerous tumor. Finally, in all suspicious cases, be careful and examine other portions of the body—the abdomen, the sub-cutaneous organs, and, perhaps, you may discover a tumor, or many tumors, which will aid you materially in the diagnosis.

The cases of cancer of the lung are too rare to enable me to present a complete history of their progress. But few cases have been recognized during life. I have succeeded once in three cases. In time, we shall understand the subject more fully. If you make yourselves well acquainted with the more common diseases with which cancer of the lungs may be confounded, and you meet with a case which presents anomalous symptoms or signs which cannot be explained, think of cancer of the lung.

The treatment of this disease is entirely palliative. If acute inflammatory symptoms supervene, they may be combated by a well-regulated antiphlogistic treatment, remembering that, as in tuberculous phthisis, you must not reduce your patient more than is absolutely necessary. In chronic cases, sedatives are the most useful remedies. A carefully regulated diet will also add materially to the comfort of the patient. But if the case is recognized during life, there is no hope of recovery, or even of considerable improvement: all that can be done is to sustain the vital powers, to calm distressing symptoms, to check complications, if possible, and to await the final event.

Ratio of the mortality from pulmonary disease in the white and colored population of Charleston during a period of five years.

White population in 1840, 13,030.		Colored population in 1840, 16,222.	
Consumption	208	Consumption	240
Pneumonia	30	Pneumonia	50
Pleurisy	3	Pleurisy	13
Bronchitis	4	Bronchitis	7
Croup	29	Croup	21
	274		331

The following statistics will illustrate the mortality from pulmonary diseases in London, during a period of eight years, 1840 to 1847 inclusive.

Total mortality, 397,871.

Phthisis, relative mortality to the whole mortality, 1 in 6·9.
- From October to April 28,242
- " April to October 28,905

Pneumonia, relative mortality, 1 in 12·7.
- From October to April 20,465
- " April to October 10,751

Pleurisy, relative mortality, 1 in 412·5.
- October to April 534
- April to October 430

Bronchitis, relative mortality, 1 in 33·4.
- October to April 8520
- April to October 3385

Laryngitis, relative mortality, 1 in 691·9.
- October to April 388
- April to October 237

Table showing, out of 100,000 persons born in London, the numbers dying from different pulmonary diseases at different periods of life:

	0 to 5 yrs.	5 to 10 yrs.	10 to 15 yrs.	15 to 20 yrs.	20 to 30 yrs.	30 to 40 yrs.
Laryngitis	19	0	2	2	1	4
Bronchitis	278	20	4	7	34	77
Pleurisy	5	6	4	0	24	21
Pneumonia	5070	244	73	54	169	217
Phthisis	1887	480	342	786	2474	2858

	40 to 50 yrs.	50 to 60 yrs.	60 to 70 yrs.	70 to 80 yrs.	80 to 90 yrs.	90 to 100 yrs.
Laryngitis.....	4	3	3	3	0	0
Bronchitis	155	317	505	354	72	5
Pleurisy	23	39	35	34	0	0
Pneumonia	367	483	426	283	50	2
Phthisis.....	2724	2136	1031	204	17	0

Of these 100,000 persons, there are alive at 5 years, 68·3 in 100; at 10 yrs. 64·9; 15 yrs. 63·5; 20 yrs. 61·6; 30 yrs. 56·6; 40 yrs. 49·8; 50 yrs. 41·3; 60 yrs. 29·8; 70 yrs. 16·3; 80 yrs. 4·5; 90 yrs. 3·6.

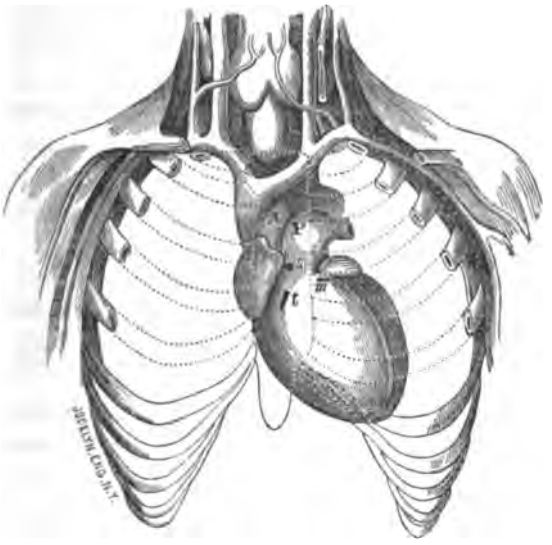
Table showing, out of 51,023 males and 48,977 females born in London, the proportion for the two sexes of different pulmonary diseases:

Laryngitis.....	22	males,	19	females.
Bronchitis	986	"	838	"
Pleurisy	111	"	55	"
Pneumonia.....	3958	"	3480	"
Phthisis.....	8297	"	6642	"

I improve this opportunity to pay a deserved compliment to Dr. A. White, the Inspector of the City of New York. His annual reports are not only much more satisfactory than those of his predecessors, but superior to those of other cities in the Union which have fallen under my notice. They compare very favorably with those of the Registrar-General of England.

I may also mention that, singularly enough, there is no annual report of the Inspector of Philadelphia, the only reports being the weekly reports in the newspapers. In conclusion, I must express my regret that I have obtained no reports from the Western cities.

DISEASES OF THE HEART.



LECTURE XXIII.

MEDICAL ANATOMY AND PHYSIOLOGY OF THE HEART.

The position of the heart in the chest and its relations to other organs.—Size of the heart: modes of measuring it during life and after death.—Sounds of the heart: their mechanism.—Situation of the orifices of the heart.—Mode of conducting the physical examination of the organ.

Before proceeding to the consideration of the individual diseases of the heart, I wish to occupy your attention with some preliminary considerations in relation to the healthy condition of this organ.* You must be well aware that the first important point in the study of all local diseases is to understand fully the condition of the organs in health. Of late years, a

* In the above sketch of the heart, the position of the sigmoid orifices of the aorta and the pulmonary artery are indicated at *s*; that of the mitral orifice at *m*; that of the tricuspid orifice at *t*. The letters A and P indicate respectively the aorta and the pulmonary artery.

branch of surgery has been much cultivated, which has received the name of surgical anatomy, the importance of which cannot be too highly estimated. Now there is a medical anatomy, which is as necessary to the formation of a correct diagnosis, as surgical anatomy is to the safe performance of surgical operations. Students often make a great mistake in this particular. They begin with the study of the diseased organ; they should first render themselves perfectly familiar with the conditions of healthy action. The proper place for you to acquire this knowledge is not the dead-house or the wards of the Hospital, but the dissecting-room, and among those who are in perfect health, by practising upon each other, for example.

In the first place, then, let me direct your attention to the position of the heart, and to the means by which it is retained *in situ*. The heart lies in the centre of the chest, inclining, however, to the left side and to the lower portion. Its direction is oblique from right to left. Superiorly, it extends as high as the fifth dorsal vertebra, and it reaches as low as the ninth vertebra. Above it are the great vessels; below, it is in contact with the diaphragm. On the right and left sides the lungs overlap it, leaving a small triangular space, but of variable dimensions, uncovered, under the cartilages of the fourth and fifth ribs of the left side. When you remove the sternum and the cartilages of the ribs and the layer of cellular tissue immediately beneath them, you expose the external or fibrous sheath of the pericardium, which is attached firmly to the diaphragm below, to the pleura on each side, to the origin of the great vessels, and to the spine above. It is this latter attachment which suspends the heart, as it were, in the chest, and which allows considerable motion laterally, and forward and backward, in obedience to the law of gravity. Under the fibrous sheet you will find the serous sac investing the heart with an external coat, and lining the inner surface of the fibrous layer. This sac is smooth, transparent, uninjected by vessels, but frequently contains a considerable quantity of clear serum. By opening it carefully, and raising the heart, you will usually find from half an ounce to two ounces of this fluid. Indeed, a much larger quantity, according to Corvisart, six or seven ounces, may exist

after a protracted agony, without being regarded as an evidence of disease. Finally, the heart itself, possessing the color, firmness, and consistence of muscular structure, is noticed with the coronary veins distinctly marked upon its surface, and with fat deposited under the pericardium, especially along the coronary vessels and towards the base of the right ventricle. On opening the organ you will usually find blood in its cavities, especially on the right side, and usually coagulated blood. Indeed, you will often notice enough to induce a considerable distension, and to render the right cavities decidedly larger than those of the left side. You will also notice, after removing the coagula, the lining membrane, smooth and transparent, resembling a serous tissue, folded into valves at the different orifices, and strengthened by an intermediate fibrous tissue. You will observe, also, the size and number of the columnæ carneæ and of the chordæ tendineæ which connect the valvular structure with the muscular tissue.

The position of the heart in reference to the anterior wall of the chest, is also a subject of practical interest, and can best be determined after the pericardium has been opened, and with a heart moderately distended by blood. The base of the ventricles will be found to correspond, nearly, with the middle of the third rib, the apex with the space between the fifth and sixth ribs, an inch or more inside the left nipple. To the right, the heart extends to the right edge of the sternum, sometimes, perhaps, beyond it; to the left, it extends nearly or quite to the left nipple.*

Various methods have been adopted to ascertain the natural size of the heart. Laennec compared it with the fist of the in-

* Hope, in the drawing he has given showing the relations of the heart to the anterior wall of the thorax, makes the organ extend two inches or more to the right of the sternum. This may happen when the right ventricle is much distended, and without being diseased. Dr. Pennock states, that the right auricle, when full, extends between the third and fourth ribs, an inch and one-third to the right of the sternum. Dr. Hope's drawing represents it still further to the right I may mention also, that Dr. Pennock thus locates the position of the ventricular septum in relation to the ribs: "The septum coincides with the osseous extremities of the third, fourth, and fifth (left) ribs; and on the fourth, is midway between the left margin of the sternum and the nipple."

dividual, and found it generally of about the same size. This is a simple and easily executed standard of comparison, but, of course, not entirely accurate. Others have weighed the heart, and found the average weight about seven or eight ounces. Bizot has adopted the still more accurate method of measuring the heart, and his examinations have been so extensive and so accurate that nothing more need be attempted on this subject. It would be well that all engaged in the careful study of the heart should adopt this method.*

Bizot's observations teach us that the heart increases in size as the periods of life advance to old age; that the size of the heart corresponds with the breadth of the shoulders, and not with the height of the individual; and that it is larger in males

* Bizot measured the heart in this way:—He first measured it as a whole—the length, by a line drawn from the apex perpendicularly to the base, to the point where the auricle joins the ventricle. At the latter point he measured the breadth of the organ, also its greatest thickness. He then opened the left ventricle, by making an incision along the blunt edge of the heart, from the apex to the base, and extending beyond the aortic orifice. He then divided the auriculo-ventricular opening, so as to change the cavity of the left ventricle into a plane surface. A line passing along the convex and adherent edges of the aortic valves, and terminating in the cut edges of the ventricle, gave the circumference of the base of the cavity of the ventricle, and a line falling perpendicularly from the summit (apex) upon the former line, gave the length of this cavity. The thickness of the parietes of the left ventricle was measured at three different points: First, near the base, six lines from the origin of the muscular fibres; secondly, at the point of the greatest thickness, which is where the third nearest the base joins the middle third; thirdly, four lines from the apex of the heart. The partition between the ventricles was measured at the same points. The preparation of the cavity of the right ventricle was somewhat different. A longitudinal incision was made from the base to the apex of this ventricle on its posterior surface, just along the line of union of the walls of the ventricle to the partition wall. A second incision was then made anteriorly, beginning at the pulmonary artery and following the same direction as the former. In this way, all that belongs to the ventricle proper was separated—the thickness of the partition wall having been already measured. The cavity of this ventricle was then measured, in the same way as that of the left ventricle. No attempt was made by this observer to measure the auricles, owing to the irregularity of their shape.

The different orifices of the heart were measured in this manner: Those of the aorta and of the pulmonary artery by a line drawn along the free edges of the sigmoid valves; those of the auriculo-ventricular orifices by a line drawn along the adherent edges of the mitral and tricuspid valves.

than in females. His observations are very accurate and numerous, being founded on the examination of one hundred and fifty-six subjects.*

In examining the cavities of the heart, it is important to ascertain, with great care, whether the orifices are of natural

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* *The following Table exhibits the Measurements of the Heart* AS A WHOLE, *at different Periods of Life, and in the Male and Female.* The measure is French, or 13 English lines to an inch.

MALES.					FEMALES.				
Age.	No. of Cases.	Length in Lines.	Breadth.	Thickness.	Age.	No. of Cases.	Length in Lines.	Breadth.	Thickness.
1–4 yrs.	7	$22^{4}/_{5}$	27	$10^{4}/_{5}$	1–4	8	$22^{5}/_{8}$	$25^{7}/_{8}$	$10^{1}/_{8}$
5–9	8	$31^{1}/_{5}$	33	$12^{4}/_{5}$	5–9	10	$26^{3}/_{5}$	29	$11^{9}/_{10}$
10–15	8	34	37	14	10–15	5	$29^{2}/_{5}$	$31^{1}/_{5}$	$12^{2}/_{5}$
16–29	18	$42^{8}/_{10}$	$45^{14}/_{10}$	$17^{7}/_{10}$	16–29	14	$38^{6}/_{7}$	$42^{9}/_{14}$	$17^{5}/_{7}$
30–49	23	$43^{3}/_{22}$	$47^{18}/_{22}$	$17^{4}/_{22}$	30–49	27	$41^{3}/_{27}$	$44^{1}/_{27}$	$14^{14}/_{27}$
50–79	19	$45^{13}/_{19}$	$52^{18}/_{19}$	$18^{3}/_{19}$	50–89	19	$42^{4}/_{19}$	$46^{11}/_{19}$	$16^{3}/_{19}$

Table showing the Size of the Heart in Relation to the Stature of the Individual.

MALES. Stature 60 in. and under. (30 cases.)

Average of Heart in lines.

Length.	Breadth.	Thickness.
$43^{8}/_{23}$	52	$17^{7}/_{23}$

MALES. Stature above 60 inches. (30 cases.)

Average of Heart in lines.

Length.	Breadth.	Thickness.
$48^{1}/_{30}$	$48^{1}/_{27}$	$17^{2}/_{27}$

FEMALES. Stature 55 in. and under. (18 cases.)

Average of Heart in lines.

Length.	Breadth.	Thickness.
$41^{4}/_{18}$	$46^{6}/_{30}$	$15^{8}/_{9}$

FEMALES. Stature above 55 inches. (34 cases.)

Average of Heart in lines.

Length.	Breadth.	Thickness.
$41^{1}/_{30}$	$43^{28}/_{30}$	$15^{4}/_{15}$

Table showing the Size of the Heart in Relation to the Breadth of the Shoulders of the Individual.

MALES. Breadth 13 inches and under.

Average of Heart in lines.

Length.	Breadth.	Thickness.
$44^{12}/_{22}$	$47^{10}/_{22}$	$16^{14}/_{22}$

MALES. Breadth above 13 inches.

Average of Heart in lines.

Length.	Breadth.	Thickness.
$45^{7}/_{18}$	$56^{7}/_{18}$	$17^{7}/_{9}$

FEMALES. Breadth 13 inches and under.

Average of Heart in lines.

Length.	Breadth.	Thickness.
89	$44^{1}/_{10}$	$17^{4}/_{5}$

FEMALES. Breadth above 13 inches.

Average of Heart in lines.

Length.	Breadth.	Thickness.
$42^{1}/_{26}$	$45^{27}/_{31}$	$14^{7}/_{31}$

size. The method proposed by Bizot is the most accurate. Another method has been suggested by Dr. Taylor, which he has found to be correct after repeated trials. The mitral orifice will just admit the first two fingers of the hand; the tricuspid orifice will just admit the three first fingers. This method is convenient, but, of course, not accurate. Another point inti-

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Measurement of the Cavities of the Ventricles, in lines.

LEFT VENTRICLE.

MALES.			FEMALES.		
Age.	Length.	Breadth.	Age.	Length.	Breadth.
1–4	20	31	1–4	$18\frac{1}{2}$	$29\frac{4}{5}$
5–9	$23\frac{1}{2}$	$35\frac{1}{2}$	5–9	$22\frac{2}{5}$	$33\frac{2}{5}$
10–15	$27\frac{2}{3}$	42	10–15	$28\frac{1}{5}$	$36\frac{4}{5}$
16–29	$33\frac{14}{19}$	$51\frac{1}{19}$	16–29	$29\frac{9}{14}$	$47\frac{4}{13}$
30–49	$29\frac{11}{22}$	$53\frac{4}{22}$	30–49	$31\frac{16}{17}$	$46\frac{4}{17}$
50–79	36	$56\frac{2}{3}$	50–79	31	$49\frac{1}{2}$
Average, from 15–79 yrs.	$34\frac{26}{61}$	$54\frac{9}{61}$	Average, from 15–89 yrs.	$31\frac{1}{20}$	$48\frac{22}{20}$

RIGHT VENTRICLE.

MALES.			FEMALES.		
Age.	Length.	Breadth.	Age.	Length.	Breadth.
1–4	$20\frac{1}{5}$	$47\frac{4}{5}$	1–4	$18\frac{7}{8}$	$44\frac{1}{8}$
5–9	24	54	5–9	$22\frac{1}{5}$	$49\frac{1}{5}$
10–15	29	63	10–15	$24\frac{2}{5}$	54
16–29	$36\frac{15}{19}$	$79\frac{5}{19}$	16–29	35	$74\frac{5}{14}$
30–49	$37\frac{12}{22}$	$83\frac{10}{22}$	30–49	$33\frac{12}{27}$	$76\frac{17}{27}$
50–79	$37\frac{1}{3}$	87	50–79	$35\frac{11}{19}$	76
Average, from 15–79 yrs.	$37\frac{14}{61}$	$82\frac{11}{60}$	Average, from 15–89 yrs.	34	$76\frac{2}{5}$

Measurement of the Thickness of the Walls of the Left Ventricle, in lines

MALES.				FEMALES.			
Age.	Base.	Middle.	Apex.	Age.	Base.	Middle.	Apex.
1–4	8	$2\frac{9}{10}$	$1\frac{9}{10}$	1–4	$2\frac{9}{16}$	$2\frac{7}{8}$	$2\frac{1}{16}$
5–9	$3\frac{1}{2}$	$3\frac{5}{6}$	$2\frac{2}{3}$	5–9	$3\frac{1}{20}$	$3\frac{1}{10}$	$2\frac{9}{10}$
10–15	$3\frac{2}{3}$	$3\frac{5}{6}$	$2\frac{1}{2}$	10–15	$3\frac{3}{10}$	$3\frac{3}{5}$	$2\frac{2}{5}$
16–29	$4\frac{2}{9}$	$3\frac{7}{9}$	$3\frac{4}{9}$	16–29	$4\frac{2}{7}$	$4\frac{7}{13}$	$3\frac{3}{26}$
30–49	$4\frac{17}{46}$	$5\frac{1}{11}$	$3\frac{12}{23}$	30–49	$4\frac{1}{9}$	$3\frac{27}{54}$	$3\frac{8}{27}$
50–79	$4\frac{27}{58}$	$5\frac{20}{58}$	$4\frac{1}{29}$	50–79	$4\frac{1}{2}$	5	$3\frac{2}{4}$
Average, from 16–79 y.	$4\frac{66}{122}$	$5\frac{10}{122}$	$3\frac{36}{122}$	Average, from 16–89 y.	$4\frac{2}{8}$	$4\frac{4}{8}$	$3\frac{13}{20}$

Measurement of the Ventricular Septum in its Middle Portion, in lines.

MALES.		FEMALES.	
Age.	Thickness.	Age.	Thickness.
1–4	$3\frac{1}{10}$	1–4	$2\frac{9}{8}$
5–9	4	5–9	$3\frac{1}{4}$
10–15	$4\frac{1}{6}$	10–15	$3\frac{4}{8}$
16–29	$4\frac{17}{18}$	16–20	$4\frac{11}{14}$
30–49	$4\frac{21}{22}$	30–49	$4\frac{11}{27}$
50–79	$5\frac{1}{2}$	50–79	$5\frac{3}{10}$

mately connected with the size of the orifices, is to ascertain whether the valves will close the orifices. I have often tried the following experiments with the healthy heart: Removing an inch or two of the aorta and pulmonary artery with the heart, and making a transverse section of the heart near the apex, so as to open the cavities of both ventricles, I then sus-

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Measurement of the Thickness of the Walls of the Right Ventricle, in lines.

MALES.				FEMALES.			
Age.	Base.	Middle.	Apex.	Age.	Base.	Middle.	Apex.
1–4	9/10	6/10	5/10	1–4	1 3/16	7/8	13/24
5–9	1 1/2	5/6	5/6	5–9	1 2/10	1	7/10
10–15	1 1/2	1 1/6	5/6	10–15	1 7/20	1 2/10	9/10
16–29	1 27/36	1 6/10	1 1/10	16–29	1 4/7	1 2/7	20/29
30–49	1 30/46	1 7/23	46/46	30–49	1 19/22	1 13/54	25/27
50–79	2 1/10	1 53/166	81/84	50–79	1 1/4	1 1/4	1
Average, from 16–79 y.	1 113/122	1 99/344	1 9/61	Average, from 15–59 y.	1 2/3	1 7/24	673/720

Measurement of the Auriculo-ventricular Orifices, in lines.

LEFT ORIFICE.

MALES.		FEMALES.	
Age.	Circumference.	Age.	Circumference.
1–4	25 1/6	1–4	26
5–9	30	5–9	27 9/10
10–15	34 2/3	10–15	31 3/4
16–29	41	16–29	38
30–49	48 9/22	30–49	40 17/36
50–79	48 12/19	50–89	44 4/19
Average, from 16–79 yrs.	45 17/30	Average, from 16–89 yrs.	41 1/3

RIGHT ORIFICE.

MALES.		FEMALES.	
Age.	Circumference.	Age.	Circumference.
1–4	29 2/5	1–4	27
5–9	34	5–9	32 9/10
10–15	39	10–15	34
16–29	50 9/19	16–29	37 12/13
30–49	54 5/22	30–49	47 4/27
50–79	57 1/3	50–89	49 13/19
Average, from 16–79 yrs.	54 12/61	Average, from 16–89 yrs.	48 1/4

Measurement in lines of the Aortic Orifice, at the free edges of the Sigmoid Valves.

MALES.			FEMALES.		
Age.	Circumference.	Thickness.	Age.	Circumference.	Thickness.
1–4	17		1–4	16 1/16	
5–9	18 1/2		5–9	17 1/8	
10–15	21 1/2		10–15	19	
16–29	26 10/19	60/100	16–29	24 3/8	80/100
30–49	30 20/22	76/100	30–49.	28 3/27	70/100
50–79	36	80/100	50–89	32 5/6	75/100
Average, from 16–79 yrs.	31 15/61		Average, from 16–89 yrs.	28 4/5	

pended the heart by hooks passed into three different points of the aorta, so as to keep this vessel open; at the same time, gently supporting the body of the heart by the hand. By pouring water into the aorta, none of the fluid should pass through the valves, which, when looked at from above, should more than close the orifice of the vessel. The same experiment uniformly succeeded with the pulmonary artery—I was also equally successful with the mitral valve. After opening the left auricle, as well as the cavity of the left ventricle by cutting off the apex, then suspending the ventricle by hooks, and supporting it gently by the hand, I have uniformly found that this valve closed the orifice, although not, I think, quite so perfectly as in the case of the aortic valves. With the tricuspid valve I have seldom or never succeeded in preventing regurgitation. The flaccidity of the walls of this ventricle may be, in part, the cause of this, but I am inclined to believe that tricuspid regurgitation is very common before death, from congestion, even when no disease of the heart exists. I may here allude to the opinion maintained by the late Dr. King, of London, that a regurgitation through the tricuspid orifice often takes place in health, and that the tricuspid valve thus becomes a safety-valve, by preventing too much blood being thrown into the pulmonary vessels.

I am surprised that Bizot, who has given us the measurements of the different orifices of the heart, did not measure the height of the valves necessary to close the orifices; yet, from the measurements he has given, you can readily calculate the proper height of the aortic and pulmonary valves, for the orifices they close form a circle. Thus, if the mean circumference of the

Measurement in lines of the Orifice of the Pulmonary Artery, taken from the corresponding point.

MALES.			FEMALES.		
Age.	Circumference.	Thickness.	Age.	Circumference.	Thickness.
1–4	$18^{3}/_{5}$		1–4	17	
5–9	$19^{2}/_{3}$		5–9	$18^{1}/_{2}$	
10–15	$22^{1}/_{3}$		10–15	$20^{2}/_{5}$	
16–29	$29^{2}/_{12}$	$^{30}/_{100}$	16–29	$28^{3}/_{14}$	$^{20}/_{100}$
30–49	$31^{13}/_{22}$	$^{31}/_{100}$	30–49	$29^{1}/_{3}$	$^{22}/_{100}$
50–79	35	$^{36}/_{100}$	50–89	$32^{17}/_{36}$	$^{26}/_{100}$
Average, from 16–79 yrs.	$32^{21}/_{61}$		Average, from 16–89 yrs.	$30^{7}/_{60}$	

pulmonary orifice is thirty-two lines, the diameter of the circle is ten lines and two-thirds, and the height of each valve should be five and two-ninth lines. The same calculation will give you the necessary height of the aortic valves—five lines. You must be careful, however, to observe, if one segment of the valve falls short of the requisite height, whether another segment does not compensate for this deficiency by an increased height. The auriculo-ventricular orifices being oval instead of circular, do not admit of this calculation with the same exactness. This is unfortunate, because the valves of the aorta and of the pulmonary artery are so irregular in their shape, that you can frequently detect any insufficiency by the eye alone, as the slightest deformity is perceptible. This is not the case with the tricuspid and the mitral valves.

When you examine the precordial region in the healthy individual, you will notice several things worthy of attention. By placing the hand over this region, you will feel the heart beating with a certain degree of force, not uniform for all individuals, but capable of being properly estimated by the comparative examination of many cases. The stethoscope, however, is a better instrument than the hand in judging the impulse of the heart. By the use of this instrument you can determine the extent of the impulse, which is something very different from the general jar communicated to the parietes of the chest, which depends more upon the force of the action of the heart, and upon the narrowness and elasticity of the thoracic parietes, than upon the size of the heart. The true impulse is felt only over the heart itself, and is a measure of its size. The shock seems to come from under the stethoscope, and this ceases the moment you leave the region of the heart, although the general shock of the parietes of the chest, more feeble and distant, may still be heard even at a great distance from the heart. The true impulse will be found to extend, above, as high as the third rib; below, to between the fifth and sixth ribs; to the right, to the middle of the sternum, sometimes beyond this point; to the left, to the nipple. You can also measure the size of the heart by the method of auscultatory percussion, introduced by Drs. Cam-

mann and Clark,* and more imperfectly by ordinary percussion over the precordial region. The natural dulness over the precordial region is by no means uniform, depending upon the relative development of the lungs, which in some cases quite cover the heart. But, usually, there is a degree of dulness over the centre of the precordial region, to the extent of about two inches, vertically and transversely; I think, also, that I can gen-

* The method of examining the heart by auscultatory percussion, published in the New York Journal of Medicine for 1840, is an important addition to our means of estimating the size of the heart during life. A solid cedar cylinder, six inches in length, and one inch in diameter, cut in the direction of the fibres, and with an ear-picce attached, is applied to the centre of the precordial region, while the ear is applied to the other end. Percussion is then made from the point near which the cylinder is applied, towards the limits of the heart in every direction. So long as percussion is made over the body of the heart, a distinct, sharp shock is felt directly in the ear; but as soon as the limits of the heart are passed, this sharp shock immediately ceases, and that even, in passing from one solid organ to another in contact with it, as from the heart to the liver. Care must be taken not to confound this shock with that more diffused shock produced by striking the ribs in any portion of the chest. A little practice will render the discrimination easy. For the purpose of accurate measurement, four diameters of the heart are taken. First, a vertical diameter, commencing at the base, just outside the origin of the aorta, and running downward parallel with the median line of the body. Secondly, a transverse diameter, which intersects the former at right angles near its centre. Thirdly, a right oblique diameter, which applies from the upper right border of the right auricle to the apex, as if drawn from the top of the right shoulder. Fourthly, a left oblique diameter, intersecting the right oblique at right angles, and ending where the right ventricle and auricle unite. Thus we obtain eight nearly equidistant points in the circumference of the heart, and can readily fill up the intervening spaces, so as to obtain the entire figure of the organ. To make a practical illustration of this method, apply the solid stethoscope over the centre of the precordia; percuss upward, just outside the origin of the aorta, until the sharp shock abruptly ceases. Mark the spot. Then percuss downward, until the shock again ceases. Mark this spot also. Connect the two points by a vertical line; this will give you the vertical diameter—and so with the rest.

By this mode of measurement, the mean diameter of the adult heart was found to be as follows:

Male.	*In.*		*Female.*	*In.*	
Vertical diameter.....	4	0 lines.	Vertical diameter.....	3	7 lines.
Transverse "	4	4 "	Transverse "	4	1 "
Right oblique "	4	10 "	Right oblique "	4	10 "
Left oblique "	3	10 "	Left oblique "	3	7 "

So that the sum of the mean diameter for the male heart is 17 inches, and for the female heart 16 inches.

erally judge of the proper dulness of this region, by the *degree* of dulness over the central portion of the precordia.

I may remark, also, that in the healthy condition of both heart and lungs, the respiratory murmur should be heard, more or less distinctly, over the whole precordial region.

In examining the healthy heart, there is no fact of more importance than that of ascertaining the point where the apex strikes the ribs. If the patient is standing, this point will be found uniformly between the fifth and sixth ribs on the left side, and generally about an inch below and to the inside of the nipple. The nipple, however, cannot be taken as a safe guide, as its position is liable to vary. You must count the ribs. It surprises me that authors have not dwelt particularly upon this fact of the position of the apex of the heart. In my experience, it is by far the most important fact among the physical conditions of the healthy heart. I shall have frequent occasion to allude to its great diagnostic value.

When you listen to the action of the heart by applying the ear to the precordial organ, you will hear two distinct sounds, each followed by an interval of silence. The one, dull, prolonged, accompanying the impulse of the heart and of course the contraction of the ventricles, is called the *systolic* sound of the heart. The other, clear, short, and abrupt, accompanying the dilatation of the ventricles, is called the *diastolic* sound. Between the first and second of these sounds there is a short, but distinct interval of silence; between the second and the first there is a longer interval. These sounds, with their intervals, constitute what is called the *tic-tac* of the heart.

The succession of the movements of the different portions of the heart, and their connection with the sounds of the organ, are now well established by experiments. The auricles contract first. This is immediately followed by the systole of the ventricles, the first sound of the heart, and the diastole of the auricles. After an interval of repose, comes the diastole of the ventricles, and with it the second sound of the heart. Then comes a longer interval of repose, after which the succession of movements and sounds is repeated.

Laennec really knew nothing of the mechanism of the sounds

of the heart. He did not even connect them correctly with the different movements of the organ, but supposed that the second sound occurred during the systole of the auricles. Mr. Turner of Edinburgh first pointed out the true connection of the sounds with the movements of the heart, by maintaining that the first sound occurred during the systole of the ventricles, and the second during their diastole. Subsequent experiments on animals by the Dublin Committee, by Dr. Hope, and by Drs. Pennock and Moore, in this country, fully establish these facts. The connection between the sounds and movements of the heart being established, an attempt has been made to explain the mechanism of these sounds. Magendie proposed a very simple theory, which, however, is not sustained by facts—that the first sound is caused solely by the striking of the heart against the ribs, during the systole of the ventricles, and the second by the same cause, during the diastole of the ventricles. But abundant experiments have proved that these sounds are heard when the sternum is removed, and every precaution used to prevent the heart from striking against any solid substance. Rouannet came nearer the truth when he maintained that the two sounds were valvular, the first caused by the tension of the auriculo-ventricular valves, the second by the tension of the sigmoid valves. The fact that the second sound is thus caused, is well established by numerous experiments, as simply pinning the valves against the sides of the vessels destroys this sound, while removing the pins at once restores the natural sound. It is much more difficult to try this experiment on the auriculo-ventricular valves, or indeed any other which shall stop the action of these valves, without interfering materially with the action of the heart in other respects. In truth, there are no experiments that I know of which establish directly the part which the auriculo-ventricular valves play in producing the first sound of the heart. Analogy, however, presents a very strong argument in their favor, for if the sigmoid valves generate a sound, why should not the auriculo-ventricular valves, which are so similar in their structure and mode of action? Indeed, I feel well satisfied that in certain cases, especially when these valves have been thickened, that I have clearly distinguished, by the ear, the valvular element of the first sound.

But if the first sound were simply valvular, it would be clear and abrupt like the second sound, instead of being dull and prolonged. That other elements of sound exist, there can be no doubt. But what are they? For myself, I cannot conceive how the heart can strike against the ribs with the force we know it does strike, without generating a sound. If I strike against the chest with the end of my finger, ever so gently, I generate a sound: why, then, should not the apex of the heart, striking the chest with equal force, also generate a sound? I believe that it does. But then observers tell you that the first sound is heard when the sternum and ribs are removed, although far more extended observations are necessary to prove to my satisfaction that the sound is not modified by this removal. There must, therefore, be some other cause for the dull and prolonged sound, independent of that caused by the striking of the heart against the walls of the chest, something, in fact, in the heart itself. Now there are two causes residing in the heart itself, which may produce this result. Dr. Wollaston has proved that muscular contraction generates a sound. This, then, may be an element of the first sound of the heart. Again, it is in accordance with the laws of physics that the rush of the blood against the walls of the ventricles, during their contraction, may generate a dull sound. This, also, may be an element of the first sound of the heart.

The sum of our knowledge of the causes of the sounds of the heart is this. The first sound is compound, and caused certainly by the tension of the auriculo-ventricular valves, and by the striking of the apex of the heart against the ribs; and probably, also, by muscular contraction, and by the friction of the blood against the walls of the ventricles.

The second sound of the heart is simple, and caused by the tension of the valves of the aorta and pulmonary artery during the diastole of the ventricles. This fact is fully proved by most abundant and conclusive experiments.*

* The student will find a detailed account of the most important experiments performed to establish the mechanism of the sounds of the heart, in the New York Journal of Medicine and Surgery, for April, 1840.

The precise situation of the different orifices* of the heart, as nearly as can be ascertained, should be distinctly recognized. The patient being in the erect position, the orifices of the aorta and of the pulmonary artery are under the centre of the sternum, and opposite the space between the cartilages of the third and fourth ribs. That of the pulmonary artery is the most superficial, and a little higher than that of the aorta. The left auriculo-ventricular orifice is under the lower edge of the cartilage of the third rib, while the right auriculo-ventricular orifice is under the sternum, opposite the cartilage of the fourth rib, and a little to the left of the median line. These facts are especially important to be remembered in the diagnosis of valvular disease.†

* In the representation of the heart at the commencement of this lecture, *s* represents the position of the sigmoid orifices; *m* that of the mitral orifice; *t* that of the tricuspid orifice; while A and P are placed upon the aorta and upon the pulmonary artery respectively.

† It is, perhaps, impossible to fix, with perfect exactness, the situation of the different orifices of the heart during life. I have adopted, very nearly, the points indicated by Dr. Hope, and I believe them to be sufficiently accurate for practical purposes. Dr. Hope places the pulmonic valves under the sternum, opposite the lower edge of the third rib, and a little to the left of the mesial line, and those of the aorta behind them, but about half an inch lower down. The auriculo-ventricular valves he places opposite the interspace between the third and fourth ribs, and the right a little lower than the left.

Dr. Pennock differs, somewhat, in his location of the valves from Dr. Hope, yet the difference is not material. "If needles be passed perpendicularly to the plane of the sternum through the middle of that bone, opposite the middle of the cartilage of the third rib, it will penetrate the *aortic valves*." "If the wires be passed perpendicular to the tangent of the curved surface of the thorax, between the cartilages of the second and thirds ribs, half an inch from the left margin of the sternum, the *semilunar valves of the pulmonary artery* are entered." "The *mitral valve* commences beneath the lower margin of the left third rib, near the junction of its cartilage with its osseous extremity (two and a half to three inches to the left of the sternum), and runs slightly downward, terminating opposite the left edge of the sternum, where it is joined by the upper margin of the cartilage of the fourth rib." "The *tricuspid valve* extends obliquely downward, from a point in the middle of the sternum, immediately below the third rib, to the right edge of the sternum, where that bone is connected with the lower margin of the cartilage of the fifth rib." In this description, the sigmoid valves are placed rather higher, and the tricuspid further to the right than in Hope's description. The situation of the mitral valve corresponds in both descriptions.

In examining the heart, it is important that the precordial region should be fairly exposed to view. In males, it is better to expose the whole chest; in females, delicacy requires you to allow a thin covering to the person; but even in them, you will add to the exactness of your examination by cautiously exposing the region of the heart. If a little tact is employed, you need not offend the most scrupulous delicacy, and yet gain all that is desired. It is important, also, to select a uniform position of the body for your examinations, as the heart is a movable organ, and the best is, undoubtedly, the erect position. Let the patient stand, with the shoulders resting against a wall, or if too feeble, let him sit in a common chair. Many patients cannot lie down in an advanced stage of heart disease without feeling oppression, and prefer the erect position. If the patient lies upon the back, the heart falls towards the spine, and the impulse, as well as the sounds, become less distinct. If he lies upon the right side, the heart falls, somewhat, in the same direction, and the relative position of the organ to the sternum and ribs is, of course, changed. In the erect position, indeed, the heart descends a little; but if all your examinations are made in this position, no error can be committed on this account.

In percussing the precordial region, the best pleximeter is the forefinger of the left hand, and the best hammer, the first two fingers of the right hand. The finger fits easily into the inequalities between the ribs. In females, and sometimes in males, the accumulation of fat about the mammary region interferes with percussion, but practice will enable you to make proper allowances for this difficulty. But I need not now enter into the details of percussion, having done this in a former lecture.

The best method of examining the sounds and the impulse of the heart, is the solid stethoscope. From comparative trials I am inclined to think that this instrument is preferable to the hollow instrument. But after all, perhaps, the best instrument is the one to which you are most accustomed. The stethoscope is indispensable in your examinations of the heart. Without it you cannot obtain a just idea of the extent of the heart's impulse, or locate with precision the seat and direction of the

sounds that may exist in the precordial region. In many cases, however, I have been able to detect by the naked ear slight murmurs over this region, which I could not hear through the stethoscope, so that it is always proper to use it as well as the stethoscope. By using the instrument you will be less confused by the murmur of respiration, which, when a soft murmur exists in the heart, may be confounded with it by an inexperienced auscultator. It is a good practice, in listening to the sounds of the heart, to request the patient to hold his breath for a moment, and then these sounds are alone heard.

In examining the heart, you should never neglect to observe whether the precordial region preserves its natural conformation. You should examine particularly the region of the neck, and observe any distension, and especially any pulsation in the veins; also the condition of the arteries, whether any increased pulsation or morbid sound exist over them. Finally, you should examine the physical condition of those organs which lie in contact with the heart—the lungs, the liver, the stomach; for physical changes in these organs may induce physical changes in the condition of the heart, independent of disease, or may modify the conditions of diseased action.

I cannot urge too strongly the necessity of studying the physical condition of the heart in the perfectly healthy individual, and of becoming perfectly familiar with all the phenomena of healthy action, before commencing the study of the diseased heart. The medical anatomy of the organ, which has formed the principal subject of my remarks to-day, and which embraces the most important facts in the diagnosis of the healthy heart, must be reviewed again and again, and accurately studied in the dissecting-room.

LECTURE XXIV.

SIGNS AND SYMPTOMS OF DISEASED HEART.

Physical signs.—Displacements of the heart.—Influence of disease on the rhythm and the sounds of the heart.—Mechanism of the blowing sound; of the friction sound.—Rational symptoms of heart disease: palpitation, pain.—Constitutional symptoms: dyspnœa: congestions, active and passive: pulse.

An appreciation of the physical signs indicating a diseased heart is, in the present state of our knowledge, quite as easy as in the case of diseased lungs. But this has not always been so. When you read the writings of Laennec, who is justly regarded as the founder of auscultation, you will be surprised at the difference in his knowledge of the physical diagnosis of cardiac and of pulmonary diseases. So that, while he is still regarded as the highest authority in the latter class of diseases, no well-informed physician thinks of consulting him in relation to the former class. I wish, at the present time, to bring before you a general view of the physical changes which disease induces in the heart, in relation to other organs in the chest, as well as in its own structure.

In speaking of the medical anatomy of the healthy heart, I have described its position and its relations to other organs. I have spoken of it as a movable organ, capable of being sensibly displaced, even by gravitation. It is also capable of being displaced by disease. There is no cause that operates so frequently or so powerfully in displacing the heart as a liquid or gaseous effusion into the cavity of the pleura, especially of the left pleura. I have frequently seen the organ pushed quite over to the right of the sternum, and that, too, without producing any marked disturbance in its functions. The same accident may result from effusion into the right pleural cavity, but the displacement is then much less. I have also described a cancerous tumor developed in the chest, which produced the same result, but these cases are rare. It may also happen that a deformity of the parietes of the chest will displace the heart,

especially a bad curvature of the spine. Disease, external to the chest, may produce the same result, as an enlargement of the left lobe of the liver, or even an accumulation of fluid, or gas, in the cavity of the peritoneum, or in the stomach. So that, in judging of the position of the heart, you must examine carefully, not only the heart itself, but the condition of the neighboring organs. The changes in the heart itself which alter its position, are liquid effusion into the pericardium, which presses it backward towards the spine, and enlargement of the heart, which tends to carry the organ downward and to the left side. In cases of very great enlargement, I have found the heart, after death, lying quite transversely in the thorax. When the right side of the organ is the seat of enlargement, it may extend far beyond the right limit of the sternum. It has appeared to me, that the tendency of the heart to enlarge upward is not so great as the tendency to extend itself in other directions. This may be owing to the fact that the base is more fixed than the other portions of the organ. In children, whose chests are usually narrow, the heart has been observed to extend upward as high as the second rib. I know of no such case in the adult.

The *true impulse* of the heart is found to increase with the size of the organ—that is to say, the extent of the impulse as measured by the stethoscope. The force of the impulse is another thing. This depends upon the muscular power of the heart, and may be permanent, as in the case of hypertrophy, or temporary, when it depends on nervous excitement. The jar which is felt over the walls of the chest, must be distinguished from the true impulse felt only over the heart itself. The former may extend to a great distance; the latter is, of course, much more limited. The impulse of the heart is best noticed at the apex, and the displacement of the apex from its natural position between the fifth and sixth ribs, is often the best guide to a displacement of the whole organ, and the best diagnostic sign of enlargement of the heart. It is usual to find the apex in the latter case beating as low as the seventh or the eighth rib, and outside the nipple; or when the heart assumes a transverse position, it may strike higher up than its natural position, and still further to the left. I am inclined to think that in cases in which

the right side of the heart is alone enlarged, the position of the apex is not much displaced. This point of the heart, indeed, may be regarded as belonging to the left ventricle alone.

The force of the impulse of the heart may be diminished as well as increased by intrinsic, as well as by remote causes. Thus a considerable effusion into the pericardial sac, will nearly annihilate all impulse, by pushing the heart backward. An advanced stage of enlargement will produce the same result, although in a less degree, by cooping up the heart, and the wearing out of its nervous energy. A soft heart, a fatty heart, an atrophied heart, also, loses its impulse. The same result may also follow from mere nervous exhaustion. I may remark, however, that nervous exhaustion is still more apt to induce the contrary condition, at least temporarily, when combined, as it usually is, with nervous excitement.

I have already stated that the impulse of the heart, both in extent and in degree, is best measured by the stethoscope, and that I prefer the solid instrument for this purpose. The limits of the healthy impulse I have also already pointed out in the preceding lecture.

Irregularity in the rhythm of the heart is not a certain indication of organic disease; it may be caused by mere nervous irritation. I have known it produced in a child from the over-action of a cathartic, and I have noticed it every now and then in typhoid fever, and in other diseases, without there being the least reason for supposing any disease of the organ.

Indeed, you will every now and then meet with individuals who have an intermittent action of the heart, either constant or occasional, who seem to be in good health, and are free from other cardiac symptoms. Yet an irregularity in the rhythm of the heart should always draw your attention to the careful examination of that organ, and you will frequently be able to discover disease. In acute pericarditis and in endocarditis, especially when severe, or complicated with other acute internal inflammation, you will be apt to notice this irregularity; also in advanced cases of enlargement of the heart. I have observed that this irregularity, which is by no means constant in the same case, is induced by opposite causes. Thus, in one patient, exercise, ex-

citement, will render the action of the heart regular, the temporary stimulus, I suppose, giving it tone, while in another case, the same cause will produce irregular action, by over-working, perhaps, an enfeebled organ. It has been thought that valvular disease is particularly apt to induce irregular action in the heart. I have not found this opinion to be true. It will more frequently be found, I think, that irregularity of rhythm is connected with a feebleness in the action of the heart from a variety of causes, and often in connection with a condition of the organ in which, at first sight, it would hardly be suspected, as in increased development of the heart, and even with hypertrophy. Whether this be owing to a radical change in the muscular fibre itself, too minute to be detected by ordinary observation, or to a tendency to softening, or to the gradual exhaustion of the vital energy from long-continued over-action, is as yet doubtful.

When the heart is pushed out of its place by effusion into the pleura, or into the pericardium, the sounds of the heart are carried with it. If *the displacement* does not alter the relative distance of the heart to the walls of the chest, the sounds are simply displaced, but not altered in their character. But if the displacing cause tends to remove the heart further from the parietes of the chest, as in effusion of liquid into the pericardium, the sounds of the heart become distant, feeble, muffled. In this case, that element of the systolic sound which depends upon the striking of the apex against the ribs, may be destroyed, but the remaining elements, residing in the heart itself, are rendered feeble, not only by increased distance, but by the interposition of a new and badly conducting medium, viz., the fluid effusion. Great hypertrophy of the heart also tends to render the sounds less distinct, and in two ways: first, the increased thickness of the walls of the heart obscure the valvular element of the sounds; and secondly, the increased size of the organ, by filling up the mediastinal spaces, leaves no room for it to spring forward against the ribs. It presses against the ribs, rather than strikes them. I have been led, from the consideration of these facts, to think that the first sound is chiefly dependent upon the causes to which I have given the chief place in the production of this sound, viz., valvular tension and the striking of the apex against

the ribs, because in cases in which this sound is very obscure and feeble, the muscular contraction may be strong, which would increase the muscular sound, as well as that produced by the friction of the blood, if, indeed, they are true elements of the first sound. Again, when the heart is dilated, and the walls thin, the first sound is loud and clear, approaching in character the second sound, although the action of the heart is feeble. In this case it is probably chiefly valvular, for the apex hardly strikes the ribs, the friction of the blood is diminished, and the muscular substance, thin and delicate, and closely applied to the walls of the chest, would more readily transmit a valvular sound from within, while any sound it might generate by its muscular contraction would be comparatively feeble. It is thus that diseased action sometimes strikingly corroborates the phenomena of healthy action.

But the sounds of the heart are not only changed in their character and in their quality by disease, they are often altered, by the addition of new elements of sound, which may coexist with the natural sounds, but more frequently mask them, or overpower them altogether. Changes in the structure of the valves may, in certain cases, indeed, produce no other change in the sounds than an alteration in their quality; and this chiefly applies to the sigmoid valves, especially to those of the aorta, and with which are connected the second sound of the heart. A slight thickening and induration of these valves seems capable of rendering the second sound of the heart more dry and crackling in its character, and, I think, heightens the tone, producing what Bouillaud has called the parchment sound. On the other hand, a thickening with softening of these valves, and especially when they are covered with soft vegetations, lowers the tone, and changes the clear, abrupt sound into a dull and muffled sound. Similar changes in the auriculo-ventricular valves may modify the first sound; but here, the difference is not so easily distinguished, owing to the complex character of this sound.

But when the valves and orifices of the heart are diseased, a new element of sound, as I have already stated, is added; and this sound, very different, in different cases, in its tone and

quality, has still one nearly constant characteristic, that of resembling a *blowing sound*—the blast of the ordinary bellows.

If you take a tube of caoutchouc, and fix to one extremity a small forcing-pump, and force through it a stream of water, no sound is heard—the tube is simply distended by the fluid as it flows onward. If, now, you tie a thread around the tube, so as to compress it equally at a certain point, and again force the fluid moderately through it, a soft, blowing sound will be heard, having its maximum over the seat of obstruction, and passing along the tube in the direction of the current. By placing the finger on the seat of the obstruction, and then carrying it along the tube in the direction of the current, you will feel a feeble thrill. If, now, you simply increase the force of the current, without altering the tube in the least, the key will be raised, and the sound become more rough, and at the same time, the thrill will become more distinct. Again, if you alter the obstruction, by rendering its inner surface rough by means of knots of thread passed into the interior of the tube, or in any other way, and apply the same degree of force with the pump as in the first experiment, the sound will be rough, and the thrill distinct, as in the second experiment in which more force only was employed. Increasing the force of the pump, will still raise the key, and render the sound still more intense. If the obstruction is great, and the force considerable, the sound may be raised to a musical note. But if, again, the obstruction is carried too far, so that the opening in the tube becomes too small, no sound or vibrations are produced; and the same is true if the obstruction is moderate, and the propelling force too weak.

This is the simple mechanism of valvular murmurs; an obstruction to the flow of blood through the orifices of the heart, which are, in fact, very short, elastic tubes, depending upon the degree and character of the obstruction, and upon the force of the action of the heart. If the obstructed orifice be smooth, and the action of the heart moderate, the sound will be soft; if the obstruction be rough, the sound will be harsh; but in both cases, the sound will be modified by the force of the action of the heart.

The cause of the sound in these cases, is the vibration of the circulating fluid at the seat of the obstruction; a vibration which is communicated to the elastic walls of the orifice, and is transmitted through, more or less, elastic media in contact with these walls.

It is a well-known fact in physics, that sounds, in travelling through different media of the same density, lose something of their intensity. This is still more striking, when the media are of different densities, and thus vary in their vibratory power. It is also a well recognized fact, that sounds generated in a moving fluid, travel in the same direction with the fluid, and hardly at all in the opposite direction.

If you will apply these facts to the physical condition of the heart, you will readily perceive that the sounds generated at the orifices of the heart will be modified according to the number and the density of the different media through which they pass. The different media are nearly the same for all the orifices. They are, first, the muscular tissue of the heart, then the different layers of the pericardium, then the loose cellular tissue of the anterior mediastinum, and finally, the sternum or the ribs. If now, by altering the position of the body, you can compress these media and bring them more closely together, you will increase the distinctness of the sounds. This is made very evident by an easy experiment. If the patient leans forward, so as to compress the different layers between the orifices and the sternum, and bring the heart nearer to the parietes of the chest, the sounds are much more distinct than when the patient lies upon the back, and the heart falls away from the sternum towards the spine. So when the stethoscope is used, which is, in fact, interposing a new medium, the sounds are less distinct than when the ear is applied directly to the chest. This is a fact important to remember. I have often recognized sounds by the naked ear, which I could not hear at all through the stethoscope. You will also generally notice, that sounds are readily transmitted along the great vessels when the current is in that direction; although there is one striking exception to this fact, which I shall notice when speaking of disease of the mitral orifice.

The obstruction of the orifices differs remarkably in the mode by which it is produced; yet the physical result is precisely the same. It may result from a narrowed orifice, by which the onward current of blood is retarded; it may also result from an imperfect closure of an orifice, by which the fluid regurgitates through the orifice. In this latter case, it is easy to perceive that the blood still flows through a narrowed orifice, and that vibrations may be generated. As, in the first instance, the obstruction may be so slight that no sound will be heard, so, in the latter case, the regurgitation may be so great, that no sound will be heard. It is a moderate obstruction to an orifice, and a moderate, regurgitating opening, that is best suited to the generation of sound. This latter fact is well illustrated in certain cases of disease in which the aortic valves suddenly give way in a mass, and nearly all the blood that has been thrown into the artery regurgitates into the ventricle, yet no sound is generated. So in cases in which the orifice is reduced to a mere chink, no sound may be heard.

There are cases in which a blowing sound, even of a high grade, is heard over the heart, and yet no obstruction of any kind exists in the orifice. *This occurs in cases of anemia and of chlorosis.* In these cases the sound is temporary, and accompanies the periods of violent action of the heart, so common in such cases. It is probable that several causes unite in producing the phenomenon in question, which takes place at the aortic orifice and during the contraction of the ventricle, and there and then only. It would seem that the increased action of the heart creates a temporary disproportion between the orifice and the ventricle, which, by its rapid, jerking contractions, forces on the blood so quickly, that the orifice is as if narrowed. But this alone is not enough to produce a bellows murmur, since simply increased action of the heart in healthy persons, as, for instance, after exercise, does not generate a murmur. Experiment seems to prove, that a thin fluid is more readily thrown into vibrations than one that is thicker; and you know that the blood in anemia and in chlorosis becomes thin and serous. Finally, the experiments of Dr. Corrigan perhaps prove, that a relaxed state of an elastic tube, from an imperfectly filled condition and want of distension of its walls, also assists in generating the vibrations of

sound. Thus, in the first experiment that I have stated with the elastic tube, the portion on the distal side of the obstructing ligature is less perfectly filled with the fluid, and consequently is less tense than the other portion, and the vibrations seem to be felt more distinctly on that account. The arteries, as well as the orifices, may be less perfectly filled in chlorosis and anemia than in health. The same explanation, viz., unfilled arteries, has been applied to the thrill and the blowing sound noticed in the arteries of the neck in certain cases of regurgitation through the aortic orifice, and to the vibrations in aneurismal tumors; but, to my mind, the explanation is not entirely satisfactory.

The blowing sound, in its higher gradations, may imitate perfectly the sound produced by a saw, or a rasp, or a file, and even ascend, in certain rare cases, to a musical note. That these sounds are all dependent on the same general mechanism, is proved, not only by experiments on elastic tubes, but by watching carefully the manner in which they alternate in the same individual case. Thus, a soft, blowing sound may become sawing, by simply increasing the force of the action of the heart, or even by altering the relation of the heart to the walls of the chest; that is, bringing it nearer to the sternum, by causing the patient to lean forward. Increased distance alone will also change a sawing sound into a soft, bellows murmur. Thus, over the valves it may be sawing, but as you pass from this region, the sound gradually becomes softer, until it finally disappears, as you recede from the region of the heart. You may also exhibit the effect of increased distance in another way. Thus, through the stethoscope the sound may be soft and blowing, while to the naked ear it becomes much more rough. The inorganic murmur of anemia seldom reaches the roughness of the sawing sound, but it may do so.

With these changes in the character of the blowing sound, it will not surprise you that it may be absent entirely, even with considerable changes in the organic structure of the orifices, and even while these changes are favorable to its production. The chief cause of these exceptions will be found in the feeble action of the heart, which may not move the current of blood with sufficient force to generate a murmur, even when the orifices are

both contracted and rough. The want of this power can only be fully appreciated, in many cases, during life; so that you may see no reason from post-mortem examination alone, or, in other words, in the actual physical condition of the heart, to explain the absence of morbid sounds. Sometimes, however, you can explain this absence by simply examining the heart; as, when you find its muscular substance very thin, or softened, or converted into fat.

Post-mortem examination teaches that the same orifices may be, at the same time, contracted, and admit of regurgitation; also that more than one orifice may be affected. You will thus easily perceive that the blowing sound may be either single or double, and when double, may have its seat at different orifices. Post-mortem examination also teaches another important fact, that the orifices of the left side of the heart are much more frequently affected by permanent organic changes than the corresponding orifices of the right side. This difference is so great as to possess a high diagnostic value in doubtful cases.*

* Bouillaud has described a singular alteration in the sounds of the heart, which I have never noticed. He mentions eleven cases in which a triple sound was heard over the heart, and one in which it was quadruple. In six cases it was the second sound of the heart that was double; in one it was the first sound; in the remainder it is not stated which was affected. In one case, in which the second sound was double, three distinct movements were felt by the hand placed over the precordia. In four cases, in which the second sound was double, three distinct blowing sounds were heard; in one case, in which the first sound was double, two distinct blowing sounds masked this first sound. In the case of quadruple sound four distinct blowing sounds were heard. In two cases there was a blowing with the first sound, and the second was double without blowing. In four cases the pulse is mentioned as being double: in two of these cases the first sound was double, in one the second sound, and in one we have no data.

Bouillaud does not offer any explanation of these curious phenomena, but states that he has never met with them except in connection with organic changes in the valves, and with contraction of the orifices of the heart. Yet in his fifth case, post-mortem examination disclosed no other change in the orifices than aortic regurgitation, there was no contraction noticed. After examining carefully these cases, it appears to me probable that the cause of this doubling of one, or of both sounds of the heart, is by no means uniform. That, first, it may be owing to a double systole of the ventricles, or rather to the systole being interrupted and divided into two contractions. Second, that it may be owing to a similar division of the diastole into two distinct portions. Thirdly, it may be owing to the valves, especially the

The vibrations which generate sound do not always generate a perceptible thrill; indeed, the thrill is the exception, not the rule. As you might expect, the thrill is chiefly observed in those cases in which the blowing sound is most intense, as when it becomes sawing. It is felt principally over the valves, and when it passes this limit, it follows the direction of the circulating current. There is one remarkable exception to this, however, as in the case of the blowing sound, that is, in disease of the left auriculo-ventricular orifice. The inorganic murmur may also be accompanied by a thrill, in certain cases.

There is another source of morbid sound, as well as thrill, over the heart, and this is the rubbing of the opposing surfaces of the pericardium against each other when coated by lymph—*the friction sound.* The idea of the friction sound is not so well conveyed to the mind by its quality, or tone, as by the sensation it conveys of a backward and forward movement. Indeed, this sound may be soft, like the lowest grade of the blowing sound, or rough and harsh, like the higher grades. It never, however, assumes a musical tone, but it sometimes becomes creaking. The distinction between the blowing and the friction sound is, as well as the accompanying thrill, sometimes so difficult as to require all the aids you can bring from collateral sources. These will be best understood when I speak of the diagnosis of individual diseases.

The friction sound, like the blowing sound, may be single or double; and when single, it may accompany either the systole or the diastole of the ventricles.* It is usually temporary, ceasing when adhesions take place.

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sigmoid, not acting on the two sides of the heart at the same moment. Thus, suppose that the valves of the aorta are thickened, their action may be sluggish, and the pulmonary valves may become tense a little the earliest, then the second sound might be doubled. But the doubling of the sounds is frequently indicated by a double blowing sound. This, indeed, seems to be the general rule. In these cases we must look to the division of the systole or of the diastole into two portions to explain the phenomena, as well as to some disease of the orifices, viz., either contraction or regurgitation.

* Dr. Taylor, of University College, London, has advanced the opinion, new to me, that the friction sound, when double, is most distinct with the ventricular diastole; and that when single it usually accompanies the diastole. On reflection,

You will, sometimes, observe a *dilatation* of the precordial region when the heart is diseased. It is a well recognized fact that internal pressure will produce a dilatation of the parietes of the chest. This dilatation of the precordial region is noticed in pericarditis with liquid effusion, in hydrops pericardii, and in cases of enlargement of the heart. Care, however, must be taken not to confound these cases with a dilatation produced by deformity of the spine, by an unusual development of the pectoral muscles, by emphysema of the lungs, or by a change in the shape of the ribs themselves. When dependent upon disease of the heart, you may always expect to find it associated with increased dulness in the same region, and, usually, with an absence of the respiratory murmur. In the case of emphysema, the natural dulness over the precordia would be diminished. It is also important to remark, that when the deformity is in the ribs themselves, the intercostal spaces are depressed, instead of being pushed out, as they are by internal pressure.*

Sometimes, in looking at the precordial region, you will observe a *fluctuating movement* there; or it may be felt by the touch. This has been supposed to indicate liquid effusion into the pericardium. But it occurs in other cases, as when adhesions of the pericardium exist, and in simple enlargement of the heart. It is far from being a constant phenomenon in any of these forms of disease. In the two latter instances, it is, per-

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it is easy to perceive why this sound should be quite as distinct, to say the least, with the diastole of the ventricles as with their systole. Experiments on the movements of the heart when exposed in animals, prove that the organ not only moves forward, but that it turns partially upon itself, from right to left, during the systole, and of course turns back again during the diastole of the ventricles. It is this kind of spiral movement that causes the friction of the opposing surfaces of the pericardium, not the forward movement. But why the friction sound should be more distinct or more frequent during the diastole I cannot perceive. In reviewing Dr. Hope's experiments, I find it stated that the ventricle appeared more elevated during the diastole than during the systole. If it should be proved that the active expansion during the diastole is greater than the contraction during the systole, it would explain Dr. Taylor's position.

* M. Woillez, a writer on the inspection and mensuration of the chest as aids to auscultation and percussion in the diagnosis of thoracic disease, states, that in 100 individuals in whom the heart was in its normal condition, 26 presented a dilatation over the precordial region.

haps, the approximation of the heart to the parietes of the chest which contributes to its production.

Now and then, on applying the ear to the precordia, a metallic ring is noticed with the impulse. This is not in the heart, but in the ear of the listener. It occurs in cases in which the impulse against the ribs is violent and abrupt.

I may notice that a sibilant rhonchus in the lung covering the heart may be broken up, so to speak, by the impulse, so as to resemble, in the time of its occurrence, a sound generated in the heart itself. If you cause the patient to hold his breath for a moment, or to cough, this sound will disappear entirely. I have observed the same breaking up of the inspiratory murmur into distinct portions, in that part of the chest near the heart, from violent action of that organ.

The *rational symptoms*, the derangements of function in the heart, are few in number, often trifling in degree, and in no way characteristic. They frequently present themselves when no real disease of the heart exists, in the opposite states of plethora or of anemia, or as the consequence of some remote irritation in the system. In many cases, also, the mere functional derangements of the heart are quite as distressing as those dependent upon organic disease. But there is this great difference. The one is, usually, a curable affection, the other seldom admits of more than an alleviation.

A person in health is not sensible of the action of his heart, under ordinary circumstances. Violent exercise, even while it induces some dyspnœa, does not, in general, induce palpitation. Most healthy persons, when they lie in bed and on the left side, are sensible of the action of the heart, if the attention is drawn to it; but in any other position, they cannot perceive it. I understand by palpitation, the action of the heart becoming sensible to the patient, in circumstances in which it is not commonly sensible. This sensation does not, always, imply increased force in the action of the heart, for this action may be feeble, and the sensation a mere flutter, accompanied, indeed, by accelerated action. This sensation may be nearly constant, or be perceived uniformly under the influence of the same exciting causes, or it may occur only at longer and more irregular intervals, and when

the exciting causes cannot readily be detected. The most common exciting cause of palpitation in those disposed to it, is exercise, especially in going up an ascent; but mental excitement has a similar influence, also, various remote sources of irritation, whether they be seated in the stomach, or in the spine, or caused by a more general disturbance of the nervous system, or by a fulness of the circulating system. Among the permanent causes of palpitation I may notice the inflammatory affections of the heart, pericarditis and endocarditis; and their consequences, adhesions and valvular disease; permanent obstruction to the circulation from organic disease in remote organs, as in the coats of the arteries, in the lungs, or in other organs. Thus, palpitation indicates an irritable heart, and this may be the result of mere nervous disorder, or of permanent organic disease, inducing an obstruction to the circulation; and between the two, are the inflammatory affections of the heart itself, which may be perfectly removed by treatment, or may leave behind them either simple nervous excitement, or, more commonly, incurable organic obstruction to the circulation.

Pain in the precordial region is another rational symptom of cardiac disease, and also it is a symptom of mere functional disturbance of the organ. It is not always pain that is complained of, but a sensation of uneasiness, or of weariness, or of fulness, or of oppression in the precordia. Sometimes it is a dreadful sensation of sinking and of exhaustion, as in cases of angina pectoris. At other times, the pain is lancinating; and in inflammation of the heart, it may be acute and sharp, or dull and obscure. In some cases, you can readily explain the cause of pain by the existence of an inflammatory affection, or by the overloaded and obstructed condition of the heart. In many cases, it is evidently external, having its seat in the intercostal nerves, and is accompanied by much external tenderness to pressure. In its most severe form, as it exists sometimes in angina, in which the patient exclaims that his heart is grasped and torn as if by iron claws, with shooting pains down the arm, no satisfactory explanation of the pain has been as yet given, although it is, probably, a neuralgia of the organic nerves of the heart propagated to the spinal nerves.

The existence of pain in the precordial region does not, necessarily, indicate either the existence or the non-existence of organic disease; neither is its absence any more significant. Like palpitation, it seems to be incidental to both organic and inorganic affections of the heart, to be frequently absent, to vary both in degree and character, without apparent cause, and to be only diagnostic, and that only in a slight degree, when associated with other and less equivocal signs of disease. The very great frequency of mere functional disturbances of the heart, and the great resemblance of their rational symptoms to those of organic disease, should be carefully remembered.

Among the *constitutional symptoms* of disease of the heart, there is none so constant or so prominent as *dyspnœa*. The causes of this symptom are very different in different cases; but in all cases it may be ultimately attributed to this—an impediment to the oxygenation of the blood. In health, the means of oxygenation are proportioned to the circulation of the blood through the lungs, and to the wants of the general system. If the circulation is accidentally quickened, as by active exercise, a temporary dyspnœa is induced. Thus in many cases of disease, in which the heart and lungs are both healthy, but in which the action of the heart is more rapid than natural, dyspnœa will ensue, as in cases of fever and of anemia. But in the case of disease of the heart, a permanent cause exists in the enlargement of the heart, and in an obstructed flow of blood through this organ, by which, on the one hand, its passage through the lungs is too rapid for the ready action of these organs, or, on the other hand, it is retarded in its passage. In either case, the blood is oxygenated with difficulty, and dyspnœa ensues. The stimulus of inflammatory action, by quickening the movements of the heart, or the consequences of this action, by obstructing the circulation, lead to the same result. Indeed, the two causes are frequently combined. Perhaps the most marked and uncontrollable dyspnœa occurs in cases when the right ventricle is hypertrophied, thus tending to a more rapid flow of blood into the lungs, while the left auriculo-ventricular orifice is contracted, preventing a return of blood from the lungs. A permanent dyspnœa, almost always, attends the different forms

of organic disease of the heart; but this is liable to be aggravated by temporary excitement causing increased action, or by temporary exhaustion causing increased obstruction. It is also increased by causes that directly obstruct the lungs, as emphysema, pleuritic effusions, bronchitis; by the pressure of other viscera upon the heart or lungs, as an enlarged liver, or a distended stomach. So prominent is this symptom, that, not unfrequently, it affords the first evidence of a diseased heart to an experienced observer, or causes the cardiac disease to be mistaken for an asthmatic affection. Active congestions with a tendency to hemorrhage, passive congestions with a tendency to dropsy, are also prominent symptoms of a diseased heart. Cerebral apoplexy is the consequence of the former; anasarca, ascites, and hydrothorax follow the latter; while pulmonary apoplexy is most frequently induced by the coexistence of both forms of congestion. If you examine a large proportion of the apoplexies before the advanced period of life, you will find them connected with a diseased heart. The connection of dropsy with diseased heart is not so uniform. Many causes conspire to produce it. Indeed, simple chlorosis may be followed by it; but when dropsical effusion commences in the lower extremities and extends gradually to other parts, your attention should always be strongly drawn to the condition of the heart.

As a general rule, the pulse is accelerated in the diseases of the heart. When the disease is inflammatory, this acceleration may be attributed to inflammation. When enlargement of the heart exists, the increased activity of the heart's action tends to quicken the pulse in the earlier stage; but the increased irritability of the organ and of the system in the advanced stage, tends to the same result. In some cases, however, the pulse is unnaturally slow, and, not unfrequently, it is irregular. Irregularity of pulse, like irregularity in the rhythm of the heart, with which it is associated, is not peculiar to any form of organic disease of the heart, but occurs, occasionally, in them all. The pulse will sometimes intermit, when the action of the heart is regular, owing to certain pulsations of the heart not being strong enough to reach the wrist. Again, a double pulse has been noticed in some of those rare cases, in which the sounds of the

heart are triple, or quadruple, and where the contraction of the left ventricle seems to be divided into two. Finally, the pulse may have a rebound, or a sensible falling back, so to speak. This has been noticed in cases in which there is imperfect closure of the aortic or of the left auriculo-ventricular orifices. I am not aware that the condition of the heart ever communicates a distinct thrill to the pulse, as has happened in marked cases of anemia, although in that case it is usually confined to the arteries of the neck.

A full and strong pulse has been supposed to belong to hypertrophy of the left ventricle, and this is often true. The pulse may be full, yet weak, in dilatation of this ventricle; it may possess the same characters in simple anemia. A small and feeble pulse, the heart acting forcibly, sometimes attends a contraction of the aortic orifice, and this is remarkably the case in regurgitation through the left auriculo-ventricular orifice. A small and feeble pulse is also apt to occur in the advanced stage of all diseases of the heart, when dissolution is threatened; also in the fatty heart. But after all, no condition of the pulse can be regarded as strictly diagnostic.

The constitutional symptoms vary with the kind and stage of the disease. As a general rule, emaciation and general debility are not very prominent, certainly not very early symptoms in heart diseases. Of the different secretions, the urine is, most frequently, affected, becoming scanty, high-colored, and with a lateritious sediment. In the strictly inflammatory affections of the heart, the constitutional symptoms are, in general, those which attend inflammation of other important organs.

The various signs and symptoms of cardiac disease will again find a place, when I speak of the history of individual diseases. I have preferred to bring before you this general review, in order that I might explain their mechanism and their leading features. Thus, in returning to them again, their nature and value will be more easily understood, and attention will not be withdrawn from the important facts of the disease, by long, and perhaps, difficult explanations.

LECTURE XXV.

PERICARDITIS.

Patholcgical anatomy; effusion of serum, lymph, pus.—Causes; influence of acute articular rheumatism; of Bright's disease.—Mortality.—Remote influences on the muscular structure of the heart.

The heart is invested, as you are well aware, by two membranes. The external, or fibrous membrane, is spread over the precordial region like a sheet, and is attached to the diaphragm below, to the pleuræ on each side, and to the origin of the great vessels above, from which it passes backward and is closely attached to the spinal column. It is this firm and strong membrane which attaches the heart to its natural situation. Above, the attachment is so firm and intimate as to allow of little movement to the base of the organ, while laterally and inferiorly, the connection is such as to allow an easy and considerable displacement of the heart, by a change in the position of the patient, or from internal pressure on the organ. This fibrous membrane possesses very little interest, in a pathological point of view. It is, no doubt, subject to inflammation, like other fibrous tissues, but like them, it leaves very indistinct traces of its existence after death. The reverse of this is true of the second, or serous membrane, the sac which is reflected over the heart so as to form its external coat, as well as over the fibrous membrane so as to form its internal investment. This membrane is very frequently the seat of inflammation, which constitutes the disease known as pericarditis.

Pericarditis is a disease of so frequent occurrence, that it must have occasionally been observed even when post-mortem examinations were much more rare than at the present day. But it then attracted very little attention, for its diagnosis was unknown. Even as late as the days of Corvisart and Laennec, this important disease generally escaped observation during life. These excellent observers and pathologists frequently

opened bodies in which the most decided evidences of pericarditis were found, and which had entirely escaped notice during life. You must not, however, infer from this remark, that no cases occurred in which the disease was recognized during life. Then, as now, the indications of acute inflammation attacking the heart were, sometimes, so direct and unequivocal, that the most ordinary observer must have been aware of their existence. But beyond the general fact, that inflammation of the heart existed, nothing was known. Its precise seat was never recognized. Even now, it is no uncommon thing for physicians to find themselves in the same predicament with the older physicians. We frequently open bodies in which the evidences of pericarditis exist, and this is the first knowledge we have of its existence. Laennec and Corvisart might regret their limited means of diagnosis, but we have no such excuse. It is, usually, inattention alone which has allowed the disease to escape us, for the means of diagnosis are now so numerous and so well established, that every educated physician should readily detect its existence.

The evidences of pericarditis found after death, are such as are noticed in all cases of inflamed serous sacs, modified a little by circumstances, and especially by the movements of the heart during life. The first effect of inflammation is to render the serous membrane comparatively dry, and to alter the natural secretion, so as to give it, when rubbed by the finger, a sticky feel, which the French writers, with their usual appositeness, have called *poissoneux*. It is the sticky sensation you will feel when handling fish which have been for some time out of the water. This condition is, however, seldom noticed after death, for it belongs to the incipient disease. An effusion of serum soon occurs, which fills up, more or less, the cavity of the pericardium, compressing the heart, and especially altering its natural position, by forcing it backward towards the spine. This serous effusion may vary very much in quantity. It may be trifling in amount, it may reach the extent of one or two pints. Physical signs also indicate that it may vary considerably in amount during the course of the disease, independently of its gradual increase or diminution, as the disease advances or recedes. It

is seldom, perhaps never, found pure and transparent, as it exists naturally, but is rendered more or less turbid by another effusion which accompanies it—the effusion of lymph.

The lymph, held at first in solution by the serum, is soon separated by coagulation. It is not usual for lymph to be effused alone in serous inflammations, unless it is connected with, and caused by previous disease of the organs with which the serous membrane is connected. If, then, the substance of the heart be healthy, you should expect to find the two effusions existing together. The extent, however, of the serous and of the fibrinous effusions seems to bear no direct relation to each other—you may have evidences of a good deal of serum and of but little lymph, or of the reverse. But this I believe to be true: when the serum is more abundant, and the quantity of lymph is small, you may reasonably infer that the inflammation has been moderate, or subacute in its character. The quantity of lymph is often so considerable as to completely cover the heart with a thick membrane. In other cases, patches of lymph, varying in extent and in thickness, are found attached to the heart. But, as a general rule, the effused lymph has a tendency to extend over the heart, covering it more or less completely. The same is true of that portion of the serous sac which lines the fibrous membrane. It is equally, with that covering the heart, the seat of effused lymph.

This effused lymph is not only found adherent to the serous membrane, but portions of it are always rubbed off by the action of the heart, and are found floating in the serum. These portions are very minute, giving the serum a turbid appearance, resembling, very much, thin indian-meal gruel. By placing some of this turbid fluid in a glass vessel, the lymph soon subsides as a sediment, leaving the transparent serum above it.

The effused lymph varies more or less in its appearance in different cases. When it is abundant, and has existed for a considerable period, its appearance is very striking when the pericordial sac is opened. More or less firm in its consistence and in its adhesion to the heart, according to the duration of the disease, of a yellowish-white color and opake, it often presents a very rough and irregular surface from the action of the heart.

The appearance of the heart in some of these cases has been compared to that of a ripe pineapple, and certainly the comparison is a very striking one. At other times the roughness is less marked. It has been compared to that of the tongue of a cat, or to that produced by smearing two surfaces with butter, applying them, and then suddenly separating them. All these comparisons are just. These false membranes vary, also, very much in consistence and thickness, as well as in color, and in their adhesion to the surface of the serous membrane. They may resist a considerable force before they are torn. They may vary in thickness from the thinnest possible membrane, to the thickness of one, two, or even more lines. When thick, they may be sometimes separated into two layers: the internal, more uniform and compact, attached to the serous sac; the external, rough, loose, and irregular. The color varies, also, in different cases. When the membrane is very thin, it is often so transparent that its existence is with difficulty detected; when thick, it is opake, and of a yellowish-white hue. In some cases, the attachment of the false membrane to the parts beneath is so slight, that it can hardly be held in its position. In other cases its attachment is firm enough to require a considerable effort to remove it. The firmer the attachment, the deeper the color, the greater the consistence of the false membrane, the longer it has existed. Indeed, in the progress of its changes it may become fibrous, or even be converted into bone-like substance.

The effusions of lymph and serum are sometimes tinged by blood, constituting what has been called hemorrhagic pericarditis. The cause of this accident is not very clear. It does not seem to be connected with the degree of inflammation. Its cause is probably constitutional.

You will sometimes find pus in the cavity of the pericardium. If pure, it indicates a very high grade of inflammation; if mixed with serum, it may be the result of the long continuance of the disease.

The appearance of the serous membrane itself, varies in different cases of pericarditis. It is not uncommon to find this membrane preserving its natural appearance, even directly under the portions of lymph that have been removed. In other cases

it is injected by red lines, or points, or small spots, which have their seat in the subserous cellular tissue. This is the characteristic inflammatory injection of serous membranes. In some cases the membrane has lost, somewhat, its natural transparency, or it may be thickened, and then it is usually more decidedly opake. Inflammation does not appear to produce any material softening of the serous tissues. It acts rather upon the subserous cellular tissue, by softening that, so that the serous membrane is more easily removed. This can be proved by tearing it with the forceps, not by scraping it with the scalpel. Sometimes, also, you will find the fibrous layer and the cellular tissue external to it, slightly injected.

It is the tendency of the serous effusion to be absorbed, either by the efforts of nature or of art. It is the tendency of the lymph to remain, notwithstanding all the efforts of art, to become organized, and to produce adhesions, which may become the cause, according to circumstances, either of subsequent enlargement, or of atrophy, or of fatty degeneration of the heart.

There is no reason to doubt, I think, that the effused lymph is sometimes absorbed, and that the pericardium regains, in all respects, its natural condition. But generally, after an attack, when the patient dies of some other disease, you will find the pericardial sac more or less obliterated by adhesions of organized lymph. In other cases, in which the pericarditis has been partial, or imperfectly cured, you will find white patches of organized lymph on the surface of the pericardium, which, when removed by scraping them, expose the serous membrane, perhaps natural in appearance, but usually more or less thickened and opake.

In recent cases of pericarditis, the muscular substance of the heart is not often affected beyond a slight congestion, with or without softening of that portion nearest the pericardium. But in cases when adhesions have long existed, the result is different. It would seem that these adhesions sometimes aid in the production of enlargement of the heart. I say aid, because, as endocarditis also commonly exists in these cases, it is probable that this latter affection is the chief cause of the enlargement of the heart. On the contrary, in cases in which the adhesions are thick and

abundant, you may find the heart actually atrophied instead of being enlarged, and the muscular coat may also be found more or less softened, and changed in color, being sometimes of a pale yellowish tint, perhaps from fatty degeneration.

A form of pericarditis has been described which has been regarded as essentially chronic, in which the effusion of serum is proportionately large, and in which the lymph, trifling in amount, shows but little tendency to become organized. Such cases are probably very rare.

The causes of pericarditis have of late years been made the subject of much attention, and almost all observers agree that it occurs most frequently in connection with acute articular rheumatism. The proportion of cases in which it occurs in this connection varies somewhat in different statements, but no one can read what has been published on the subject, or have observed the disease in actual practice, without being struck with the general truth of the statement. So frequently, indeed, is pericarditis associated with acute articular rheumatism, that you should never allow a patient, suffering from the latter disease, to escape repeated and careful examinations to detect its existence. And this is more especially important, as the cardiac inflammation may be latent, and only revealed by the physical signs. The same remarks are equally true of endocarditis, which is frequently associated with pericarditis, or may exist independently of it.

The frequency with which pericarditis occurs in acute articular rheumatism, appears to depend somewhat upon the severity of the rheumatic disease, the number of joints affected, and the degree of constitutional fever. It has also been observed to occur most frequently with the first attack of acute rheumatism, which is also, usually, the most severe attack. But it is by no means confined to these cases. It is met with in cases of subacute articular rheumatism, in which the local affection of the joints is slight, and the constitutional excitement is moderate. The disease is very rarely associated with cases of rheumatism which may be called chronic, or in which the acute symptoms have subsided. I have never known it occur in connection with simple muscular rheumatism.

It has been common to explain the affection of the pericardium in acute rheumatism by the doctrine of metastasis. But this view of the subject will not bear a careful examination. It is true that cases are occasionally met with, in which the inflammation suddenly leaves the joints to attack the heart, but these cases are rare. Generally, the heart affection comes on after the affection of the joints, and without any mitigation of the articular inflammation. In a majority of cases this occurs early, perhaps during the first week, although it is not unusual for the heart to be attacked at a much later period. It occasionally happens that the heart is attacked first, and the joints subsequently, or the affection of the joints is so trifling as almost to escape observation, bearing no proportion to the severity of the cardiac affection. So that after a careful study of the subject, you must, I think, be brought to the conclusion that the cardiac inflammation, as well as that of the joints, are equally the result of a common cause, which may be in the blood, or in some unknown condition of the system.

The study of the exciting causes of acute rheumatism tend also to confirm this idea. It is common to refer the attack to exposure to wet and cold, and there can be no doubt that this is the exciting cause in many cases: that is to say, the exposure is the reason why the attack occurred at a particular time. But in many cases you can trace the rheumatic attack to no such exposure. Besides, the disease is very common in certain individuals, while it never shows itself in others who have been much more exposed to the exciting causes, dampness and cold. Thus you are compelled to look to a constitutional cause, and this may show its effects either by attacking the joints or the heart in succession, or both at the same time.

A large proportion of the cases of rheumatic pericarditis occur before the middle period of life, simply because, as you know, acute articular rheumatism is more common before this period. The same fact is true of endocarditis, and it is to these two causes, united or single, that must be attributed a very large proportion of the cases of enlargement of the heart which occur before the age of forty-five years. I shall have occasion to allude to this fact again, and it cannot be too strongly impressed

upon your memories. It possesses a high diagnostic value. For, so rare is it to find a case of enlargement of the heart, or of valvular disease, in a comparatively young subject, without the previous occurrence of acute articular rheumatism, that the strongest evidences of their existence should be present to satisfy you of the fact. Indeed, I have been struck with the frequency of inflammation of the membranes of the heart in young children. It is not uncommon in children of three or four years of age. I have noticed it much more frequently than I had expected from the perusal of the works of authors. I have recently, however, met with some statements which confirm this impression. Dr. Macleod, in his work on rheumatism, makes the following statement: Eight cases of acute rheumatism were under fifteen years of age; of these, four had pericarditis. Ninety-four cases were between the ages of fifteen and twenty-five years; of these, thirty-two had pericarditis. While, after the age of thirty years, the proportion rapidly diminished. Thus, there were twenty-seven cases of rheumatic fever between the ages of thirty and thirty-five years, and only three had pericarditis. Again: a still more striking statement follows. There were twenty-seven cases of rheumatism between the ages of thirty-five and forty years, and no pericarditis in any of them. This striking table of Dr. Macleod may not be fully sustained by the general experience of the profession. I think it will not. But it establishes the fact of the frequent occurrence of pericarditis in connection with acute rheumatism in young subjects. By referring to Rilliet and Barthez, I find the same fact confirmed. Thus, in eleven cases of articular rheumatism, between the ages of one and fifteen years, four had acute pericarditis.

The frequency of pericarditis in connection with acute rheumatism, has been studied by the numerical method of M. Louis, and by no one with more attention than by Dr. Taylor, as published in the Medico-Chirurgical Transactions. This observer states, that in seventy-five cases of acute, or of subacute, rheumatism, treated by him at the University College Hospital, London, six had pericarditis of considerable severity; two, in a very slight degree; and in two, its existence was doubtful:

thus making the proportion about one in eight or nine cases. Dr. Budd, of King's College Hospital, London, makes the following statement: In forty-three cases of acute rheumatism, pericarditis occurred five times, or about one in eight or nine cases. Chomel is the only author of established reputation who has found the ratio much lower than this. He states (I take the statement from Dr. Taylor) that in forty-nine cases of articular rheumatism, carefully observed, no example was found of pericarditis, or of endocarditis. Again: in thirty-one cases of acute rheumatism, only one patient had symptoms of disease of the heart (palpitations), and in one case only, the rheumatism was complicated with disease of the heart, beginning while the patient was in the Hospital. The blowing sound was heard in seven cases, and in two others its presence was suspected only.

On the other hand, Bouillaud has stated in the first edition of his work on the Heart, that pericarditis would be found in, at least, one-half the cases of acute rheumatism. In the second edition of this work, published after an interval of six years, the author still adheres to this general statement, which is, however, unsupported by statistical calculations in relation to pericarditis. He makes, however, the following statement: In one hundred and fourteen cases of acute articular rheumatism, seventy-four were of a great or of medium intensity, and forty were mild cases. In the seventy-four more severe cases, endocarditis, or endo-pericarditis, occurred sixty-four times; while in the forty milder cases, these complications only occurred a single time.

These conflicting statements must teach you that statistics when applied to medicine do not always lead to uniform results. Neither is this to be expected. Many circumstances, even supposing the observer to be unprejudiced and capable, which is far from being always the case, are likely to alter the general result. Certain complications may be more frequent in one season, or in one place than in another, and something, at least in Hospital practice, must be allowed for accidental circumstances attending the admission of patients. Every Hospital physician, I think, must have noticed that he sometimes encounters an unusually large or small number of a certain class of

cases, and can refer the difference to nothing but the mere accident of their admission. My own impression, founded on the experience of this Hospital, is, that pericarditis does not occur as often as once in every eight or ten cases, as recorded by Drs. Taylor and Budd, while it is more frequent than the statements of Chomel would seem to imply, although he does not carefully distinguish rheumatic pericarditis from endocarditis.

Pericarditis occasionally occurs in the course of other febrile diseases besides rheumatism. It is met with in scarlatina, in measles, and in small-pox; also in typhus fever, and in erysipelas.

Dr. Taylor, whose excellent paper on pericarditis I have already quoted, has established the frequent association of this disease with Bright's disease of the kidney. In thirty-one patients affected with acute pericarditis, nine, if not eleven, had Bright's disease, and no other cause of the pericarditis could be discovered. In fifty post-mortem examinations of patients who had died of Bright's disease, or were found after death to have this disease in a somewhat advanced stage, five, or one in ten, had acute pericarditis. Thus, if the statements of Dr. Taylor are confirmed by subsequent observation, Bright's disease is nearly as frequently associated with pericarditis as acute articular rheumatism. But there is this essential difference. The rheumatic pericarditis, occurring in comparatively healthy subjects, is generally cured, or at least terminates by adhesions; while that occurring in Bright's disease, a chronic affection by which the vital powers are exhausted, usually terminates fatally.

But while you recognize the frequent association of pericarditis with rheumatism, and with Bright's disease, you must not expect to find all the cases thus associated. Cases occur in which no such cause can be discovered. Like pneumonia and pleurisy, inflammations with which it is frequently associated, its cause will escape you. In some cases, it would appear that the pericarditis has followed pleurisy by an extension of the inflammation from a neighboring tissue. But in other cases it is difficult to determine the priority in the two inflammations; they probably commenced at the same time, and under the influence

of the same general cause. Indeed, the frequent coexistence of these different inflammations of the chest in acute rheumatism, as well as in Bright's disease, might lead you to suppose a common origin. But pericarditis, like other inflammations, is frequently spontaneous—no probable cause can be ascertained.

The mortality of pericarditis, generally considered, is not very great.* Most of the cases that occur in connection with acute rheumatism recover from the immediate attack, as, also, do the cases in which there is no evident cause for the disease. Those cases, on the other hand, which occur in connection with Bright's disease, also in scarlatina and in other fevers, are much more fatal. Probably a large majority of such cases terminate fatally. You would naturally expect, also, that those cases which are associated with pleurisy, or with pneumonia—and these complications are of very frequent occurrence—would often have the same fatal termination. Very severe cases, in which pus is effused into the pericardial sac, probably all terminate fatally.

If the danger of pericarditis is not usually immediate, is it so prospectively? It is the general opinion, I believe, that the adhesions which so frequently take place may embarrass the action of the heart, and lay the foundation of its subsequent enlargement. But these adhesions are usually associated with the consequences of endocarditis, and it is difficult to assign the part

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* The report of the City Inspector of New York during three successive years gives 108 deaths from inflammation of the heart; one in 425 of the general mortality. These were probably all cases of endocarditis and of pericarditis, no attempt having been made to distinguish them in the Report. Of these cases, 49 were males, and 59 were females. Forty-four cases occurred from June to December, 64 from December to June. Nineteen cases were under 10 years of age, 56 cases between 10 and 40 years of age; 33 cases were past 40 years of age.

The relative mortality from pericarditis to the whole mortality in London, during a period of eight years, 1840 to 1847 inclusive, is 704·2.

From October to April.....295 deaths.
" April to October.....270 "

Of 100,000 persons born in London, the ratio of deaths from pericarditis, and at different ages, is the following:

1 to 5 years, 7; 5 to 10 years, 2; 10 to 15 years, 6; 15 to 20 years, 8; 20 to 30 years, 8; 30 to 40 years, 21; 40 to 50 years, 7; 50 to 60 years, 12; 60 to 70 years, 3.

Of 51,023 males born in London, 32 die of pericarditis.
Of 48,977 females " " 19 " "

which each performs in altering the size of the heart. My own impression is, that endocarditis is by far the most common, as well as the most direct cause of enlargement of the heart; but that pericarditis, by producing adhesions, should lead to the same result occasionally, is, I think, very probable. I have seen pericarditis remotely fatal, not by inducing enlargement of the heart, but by inclosing it in a complete case like bone, from the changes of the false membranes, and producing atrophy of the muscular structure of the organ.

The duration of simple pericarditis is from two to three weeks. In six cases, recorded by Louis, the mean duration was fifteen days. In four other cases, resolution commenced about the thirteenth day, and the period of cure was fixed at the seventeenth or eighteenth day. In cases when complications exist, the duration is longer. In four cases resolution commenced on the seventeenth day, as a mean, but the cure was not complete until the fifty-fifth day.

LECTURE XXVI.

PERICARDITIS.

Symptoms; illustrative cases.—Appreciation of the rational symptoms, the physical signs, the constitutional symptoms.—Chronic pericarditis.—Diagnosis.

Acute pericarditis, in its primitive form, is usually ushered in by constitutional symptoms of moderate intensity. It is often a subacute, rather than an acute inflammatory disease. The patient is attacked by chills, followed by febrile reaction, an accelerated pulse, but regular; a tendency to palpitation, dyspnœa, pain in the precordial region, often slight, sometimes absent; and a trifling dry cough. The countenance is pale, and not expressive of much suffering. The appetite is impaired, there is thirst, a coated tongue, restlessness, as in other forms of inflammatory disease. If you examine the precordial region at the commencement of the attack, a friction sound may be de-

tected, but it may soon cease, and you will then notice that the natural dulness over this region is increased, both in extent and in degree, that the respiratory murmur is absent, that percussion is often painful. If you apply your hand to this region, you may not perceive at all the impulse of the heart. If you listen to the sounds of the heart, they are heard; but they are distant and indistinct. Sometimes there is dilatation over the precordial region. These symptoms and physical signs indicate a pericarditis of moderate intensity, in which a considerable quantity of serum with comparatively little lymph, has been effused into the pericardial sac. After a time, the febrile symptoms are subdued, the respiration is less oppressed, the palpitation diminishes, the pulse becomes less frequent; and with this improvement of the rational and of the constitutional symptoms, the dulness on percussion diminishes, the sounds of the heart become more superficial and distinct. While the liquid effusion is being absorbed, you will sometimes notice that its quantity seems to vary at different times, but that still the general progress of absorption continues, until, at length, a rubbing sound, often slight and transient as at the commencement of the attack, may be heard over the heart, as the false membranes on the opposing surfaces of the pericardium are brought in contact, and before adhesion of these surfaces has taken place. The time necessary for these changes is from two to three weeks.

In other cases, the disease is more active. There is a greater effusion of lymph. The friction sounds which may have existed at the commencement of the subacute form of the disease, but soon gave place to the signs of simple serous effusion, continue in this form of the disease. At the same time, there are evidences of considerable serous effusion. There is extended dulness over the precordial region, perhaps dilatation; the respiratory murmur is not heard there, but there is a friction sound, usually double, heard sometimes most distinctly at the base of the heart, sometimes under the cartilage of the fourth rib, sometimes at the apex. The action of the heart is tumultuous, but not very distinct, and a thrill can sometimes be detected as the hand is applied to this region. At the same time there is palpitation, and precordial pain, and dyspnœa, all increased by

whatever excites the circulation; anxiety, and sometimes a tendency to syncope. Sometimes there is an irregular pulse, sometimes œdema. There is considerable constitutional excitement. In the progress of the case, the friction sounds may cease for a time if the serous effusion is much increased, but they often continue until this effusion is absorbed and adhesions take place. The heart, in this more acute form of the disease, is apt to be left in an irritable condition, even after all inflammatory symptoms have subsided.

There are cases in which the disease is still more acute; cases in which pus rather than lymph is secreted by the pericardium. These cases are marked by constitutional and by rational symptoms of the most intense character. Great constitutional excitement, with anxiety and a tendency to syncope, exist, with an irregular pulse. There is palpitation, sometimes atrocious pain in the precordial region, dyspnœa, inability to move without the greatest distress, especially to the local heart symptoms. And yet, with all this evidence of severe disease, the physical signs may not be very distinct. You may discover but little dulness on percussion; the action of the heart is superficial; the friction sounds may be indistinct, or absent. The effusion is moderate in quantity, but it is chiefly pus. Death usually occurs in five or six days from the attack.

The same aggravated constitutional symptoms may be noticed in cases in which a pleurisy or a pneumonia complicates the pericarditis. In this case, the rational symptoms may be indistinct, or even latent, while the physical signs are well marked. When pericarditis occurs in connection with acute articular rheumatism, it may be still more latent, for the striking constitutional symptoms—dyspnœa, a tendency to syncope, and irregular pulse—are not present. The rational symptoms, also—pain and palpitation—may be very indistinct, but the physical signs are usually well marked.

I will relate a case which occurred in this Hospital many years ago, and which was recorded by Dr. A. Clark, then resident physician. In this case the effusion of serum was enormous. It will, therefore, serve to illustrate the physical phenomena of this condition.

An Irish laborer, aged thirty years, large and well developed, entered the New York Hospital, January 16, 1832.

He had been attacked three weeks before, by the symptoms of acute inflammation of the chest, which had been treated by venesection, purgatives, &c. He presents recent scarifications over the epigastrium, and has a recent blister over the anterior part of the chest, and on the abdomen.

The patient is pale, feeble, and œdematous. The pulse is 130 to 140 in a minute, irregular, weak, and, after any exertion, fluttering. There is cough, attended by slight mucous expectoration. The respiration is oppressed, and this is increased by the slightest exertion. The tongue is slightly coated, and the bowels are constipated. There is nausea and vomiting.

On examining the precordial region, there is marked dulness on percussion, and the dulness extends to the corresponding region on the right side. This dull portion is limited, superiorly, by a line slightly curved upward, and extending from one nipple to the other; laterally, by lines from the nipples, and diverging a little to the margin of the ribs. If the patient be placed on the right side, the dulness extends nearly an inch further to the right, and recedes from the left side, and vice versa. Not the slightest respiratory murmur can be heard over this dull region, although it is distinct in the other portions of the chest, mixed with a little sibilant rhonchus, or mucous rattle. The impulse of the heart is not considerable, but can be perceived all over the dull region; when the patient is more calm, the rhythm is perfect, and the sounds appear normal, and are most distinctly heard at the base of the organ. The apex does not strike against the ribs in any position of the body, not even when the patient lies upon his abdomen. A slight undulatory motion is observable in the epigastrium, and along the anterior part of the lower intercostal spaces. The action of the ribs, in respiration, is not considerable.

The patient was ordered a purgative, followed by a pill of calomel, squills, and digitalis, and allowed a moderately full diet, for which he had some appetite. Salivation was soon induced, but the urine was not increased. The patient gradually improved in strength, so as to sit up for a considerable time.

On the 27th day after admission, the ptyalism had gradually disappeared. A trifling diarrhœa, which had existed, had ceased. The œdema had nearly disappeared, and given place to evident emaciation. The appetite was tolerably good, the pulse regular, and it had some firmness. The patient sits up during the day, and sleeps well at night. The dulness and other physical signs continue as at first noticed. The cough and some dyspnœa also continue. Two blisters have been applied to the precordial region, and dry cups.

The patient used no medicine for the next seventeen days, and his condition remained about the same. He was ordered the supertartrate of potass, ʒ ii. three times a day. In a few days this was discontinued, for the patient seemed worse—diarrhœa ensued, the appetite became impaired, and the tendency to effusion seemed to increase.

A day or two after this, the 49th day after admission, the patient fainted while at stool, and imperfectly revived. Indeed, the next day the pulse was fluttering, and hardly distinguishable at times. The countenance was livid, the respiration slow, labored, and deep. The appetite was gone, the intellect unimpaired. Ammonia and wine.

He died, suddenly, the next day, fifty-one days after his admission, and about ten weeks after his attack. The body was but little emaciated, the development full, the face and neck livid. The integuments of the trunk and face were œdematous, but not those of the legs. Considerable serous effusion existed in the peritoneal cavity. On opening the thorax, the pericardium was found enormously distended and tense, occupying the whole anterior part of the chest, and it had pushed the diaphragm downward, so as to form a very large convexity towards the cavity of the abdomen. The liver was pushed downward, so that its upper convex margin reached the margin of the ribs. Both lungs were pushed into the posterior and lateral portions of the thorax. Had the entire contents of the pericardial sac been fluid, it could not have been less than ten pints; but, as it was, there was at least a gallon of clear, yellow serum. A thick deposit of lymph adhered universally to the serous surface of the pericardium, and its inferior surface was stained with blood.

The opposite surfaces were united posteriorly by bands of adhesion, and these had evidently existed anteriorly, but had been separated by the serous effusion. The evidence of this was a layer of lymph, here and there on the pericardium, and the absence of it in other extensive patches. Besides the general investment of false membrane, there were superimposed upon it patches, irregularly attached, some by their entire surface, others by their edges, and floating in the serum. The heart seemed rather large. The pericardium itself was one-eighth of an inch thick, and felt like leather.

The edges of the lungs were adherent to the pericardium on each side, and a small quantity of fluid was found in the pleural sacs; but the lungs were generally healthy. No other morbid appearances were observed.

You have in this case, perhaps, the most remarkable instance of inflammatory serous effusion into the pericardium on record. It is to be regretted that the quantity was not accurately measured. The physical signs of copious effusion were of course most distinctly marked. I am surprised, however, to find no mention made of dilatation of the parietes, which I think must have existed. It is also stated, that the impulse of the heart could be perceived all over the dull region; but it must have been the shock of the fluid, and not directly the shock of the heart, especially as it is expressly stated, that the apex, the most impulsive portion of the heart, did not strike the ribs, even in the most favorable positions. We should certainly expect to find, with such a copious effusion, the impulse of the heart null, and the sounds distant and feeble.

A physical sign was, however, noticed, which has not usually been noticed in cases of liquid effusion into the pericardium, and which, probably, does not occur unless the effusion is very considerable. A change in the position of the body, from the left to the right side, altered the limits of the dulness without diminishing its extent.

The case is more acute than most cases in which the liquid effusion predominated. Still, you must recollect, that the effusion of lymph was considerable, when considered by itself, although small, when compared with the enormous effusion of serum.

In the case which I have just related, the signs of liquid effusion predominated. I will relate another, in which the friction sounds constituted a prominent feature of the disease.

A tailor, aged thirty years, entered the New York Hospital, Feb. 7th, 1846.

The patient stated that he was taken sick two weeks ago, with pain in the anterior of the chest, dyspnœa, and vomiting, but no cough. His pulse is now 112, there is slight dyspnœa, the pain in the chest has nearly gone—no physical signs of disease in the chest.

Feb. 16th.—The patient states that he still feels a little pain in the left side of the chest, extending from behind, around to the mammary region; that he experiences a sense of oppression when he lies on the left side; and that the action of the heart is somewhat increased. It is not, however, increased by motion. His lips are noticed to be blue, and there is œdema of the feet and legs; the pulse has become irregular (112), and there is a little cough; the tongue is a little coated, but is moist.

The action of the heart is superficial. Over the precordial region there is a double friction sound, like that produced by the tearing of silk, not synchronous with either sound of the heart, extending over the base of the heart, and as far as the sternum. At the base of the right lung exists a mucous rattle, but without any modification of the respiratory murmur, which is also natural in the other portions of the chest.

℞. Calomel. Pulv. Scill., Pulv. Digit. āā. gr. i., ter. in die. C. C. ad reg. Cordis.

Feb. 17th.—The same friction sounds are heard.

Feb. 19th.—There is now a double sawing sound over the whole region of the heart, but having its maximum over the body of the left ventricle towards the base, between the cartilages of the third and fourth left ribs, and under the sternum; the morbid sounds are not distributed along the course of the aorta; that accompanying the diastole of the heart is rather more distinct than that accompanying the systole; the action of the heart is superficial, but percussion over the precordial region is dull; the apex of the heart appears to strike in nearly its natural place.

There is no precordial pain; the patient thinks that this, as well as the dyspnœa, were relieved by cupping; respiration is 28 in a minute; pulse regular, of moderate strength, 108 in a minute; skin cool; quantity of urine increased; tongue covered by a thin, white coat.

February 20th.—The patient is now standing, and the pulse is 120, soft and regular. The morbid sounds over the heart remain the same, except that they now extend to the top of the sternum. The gums became sore yesterday. Omit the pills, and use diuretic drinks.

February 23d.—The mouth continues sore. No pain in the region of the chest: respiration easy and free; pulse 96, regular and soft. The dulness over the precordial region seems somewhat diminished; the morbid sounds over the heart have very much diminished in intensity. Over the left ventricle they can hardly be heard at all; over the base of the heart they are more distinct, and now their maximum is between the third and fourth ribs, a little to the right of the sternum, and above that point.

February 26th.—The action of the heart is not increased by walking about the ward. The morbid sounds have very much diminished. Over the left ventricle they have ceased entirely. Over the base of the heart and the region of the valves they can hardly be distinguished. They are now principally confined to the lower portion of the sternum, extending over to the right side, about the cartilage of the fifth rib. The two natural sounds of the heart may be heard, distinct from the morbid sounds, which seem as if introduced between them.

February 28th.—The patient feels neither pain about the precordial region, nor dyspnœa, nor palpitation. The morbid sounds over the heart have entirely ceased.

March 5th.—Discharged cured.

I have never met with a case which illustrated better the progress of the friction sounds in pericarditis with but little serous effusion. The diffusion of the sounds, the frequent changes in their maximum point, their rubbing character, and their rapid disappearance, leave no doubt, in my mind, as to the correct diagnosis of the case.

The precise time when the pericarditis occurred is not known. When the patient entered the Hospital, he appeared to be recovering from some acute attack in the chest, and was, probably, not very carefully examined. No physical signs were detected, which proves that the chest was, however, examined. The occurrence of an irregular pulse, with œdema of the feet and lividity, led to a subsequent examination of the heart, and then the signs of pericarditis were noticed; also traces of pneumonia in a state of resolution. The duration of the disease from the time it was discovered was thirteen days.

The influence of the treatment is worthy of attention. Cupping produced no change in the physical signs; but soon after the constitutional effect of mercury was observed, a marked improvement in the condition of the heart was noticed.

I will now call your attention to a third case of primitive pericarditis, in which both the effusion of lymph and serum existed in a considerable quantity, and in which the physical signs of both these conditions were present at the same time. It is condensed from the Report of Dr. Taylor.

A girl, aged sixteen years, of rather delicate constitution and of slender frame, had been for three months affected with suppression of the menses and with chlorotic symptoms, attended by palpitations and œdema. The menses at length returned three weeks ago, and while they were present she washed some linen. The same evening, she complained of languor, rigors, and fever; the legs swelled, and she felt uncomfortable about the region of the heart.

On admission to the Hospital, the face was very pale, the countenance anxious, the skin hot and dry, the respiration hurried and labored, and the dyspnœa increased on the slightest exertion. Pain was felt in the region of the heart, and palpitation. There was discomfort in every position. The pulse was regular—140. There were startings in sleep. The tongue was moist and clean; the bowels open. A coarse crepitation, with dulness on percussion, existed at the base of the left lung. A double friction sound existed over the heart.

The patient was treated by six leeches to the precordia, which were repeated the next day; by mercurials, in combination with

sedatives, and by blisters, all of which produced no material relief, although the gums became affected.

On the eleventh day after admission, the following condition existed: The patient lay on her left side; turning on the back produces a sense of suffocation. Cough slight, with a trifling expectoration of colorless mucus. Respiration shallow and hurried—fifty in a minute. Face very pale; no œdema; no albumen in the urine. The left lung was, generally, dull on percussion, most marked in the lower half, and posteriorly. Inferiorly, there was no respiratory murmur; but a large muco-crepitant rhonchus (rattle?). Above, the respiration was puerile. In front, the respiration was not audible under the clavicle. The right lung was dull infero-posteriorly; in front, it was clear on percussion. The respiration was, generally, puerile over this lung, with some muco-crepitant rhonchus.

No dilatation over the precordial region, but percussion dull, transversely, to the right of the sternum, and beyond the left nipple; and perpendicularly, from the clavicle to the edge of the false ribs, most marked inferiorly. An undulatory movement was observed over the precordia, especially below the left nipple. A loud, double friction sound was heard over the whole region of the heart. It was most distinct and rough on the left side, and especially below the nipple. It was audible at the top of the sternum, but not above the clavicles. It was heard distinctly over every part of both sides of the chest behind, but with the heart's systole only.

The patient died suddenly three days after this, and about five weeks after the attack. The friction sounds were heard on the day of her death.

The pericardium occupied most of the anterior portion of the chest. It extended up to the clavicle, and its transverse diameter was eight inches: its figure was pyriform, the base inferiorly. The fibrous layer adhered, but loosely, to the pleura in contact with it: it was much thickened and vascular externally. Its cavity contained eight ounces of serum, deeply tinged with blood, and which deposited, after standing, a soft, gelatinous substance. The whole inner surface of the pericardium was covered by a very thick layer of soft lymph, of a deep red color,

very uneven, and projecting, here and there, in large processes, resembling a cock's comb, and inclosing large cellular spaces. There were a number of adhesions extending across the pericardium, from a quarter to three-quarters of an inch in length, and one or two lines in diameter. The heart, as a whole, was somewhat enlarged, pale, and rather soft. Vegetations existed upon the mitral valve, colorless, firm, of the size of a pin's head, and situated along the free edge on the auricular aspect. Similar granulations existed on the free edge of the aortic valves, and on the ventricular aspect. They could be removed by a little scratching. Two or three minute granulations were also noticed on the free edge and auricular aspect of the tricuspid valve. Nothing remarkable was noticed about the orifices or the cavities of the heart. A little turbid serum existed in both pleural sacs, with old and extensive adhesions of the left lung posteriorly, and especially inferiorly. Left lung small, especially the inferior lobe, which was fleshy, non-crepitant, sinking rapidly in water, but the texture was not diseased. Right lung healthy, with the exception of a little lobular pneumonia in the superior lobe. Nothing remarkable in the abdominal cavity. The head was not examined.

This case presents the different symptoms and physical signs of pericarditis, attended by both the liquid and the solid forms of effusion. The dulness on percussion in this case was, probably, more owing to the great thickness of the false membranes than to the serous effusion. Yet the latter was abundant enough (eight ounces) to increase the dulness, and still not enough to prevent the friction of the opposite surfaces. It will be recollected that the friction sound was very extensive, being heard all over the chest, but that posteriorly it was single and systolic. You may infer from this, that the systolic sound was the loudest, and may, perhaps, refer the great diffusion of the sound to several causes. First, to the universality of the effusion of lymph; secondly, to the considerable condensation of the pulmonary tissue, especially from previous pleuritic inflammation; and thirdly, to the narrowness of the chest, the patient being described as of a slender frame. There were also evidences of old inflammation, probably rheumatic, about the

valves, and a slight enlargement of the heart, which may have preceded the pericarditis, but the orifices seemed natural. I should remark, however, that when the patient was first admitted to the Hospital, a systolic blowing sound was heard at the apex, which, however, was transient, and as it is not given on the authority of the attending physician, I have omitted it in the statement of the case.

Finally, an undulatory movement was noticed in the precordial region. In this case it may have been caused by the liquid effusion. But in many other similar cases this is not noticed.

I have remarked that pericarditis is sometimes rapidly fatal, with the secretion of pus, and without well-marked physical signs. The case which I shall now relate is a well-marked instance of this form of the disease. It is also a remarkable case of rheumatic metastasis.

A baker, aged thirty-one years, was attacked with severe acute articular rheumatism. He was bled five times, to the extent of twelve ounces each time, and had forty leeches applied to the affected joints before much relief was experienced. This treatment was commenced about the fifteenth day from the attack, and continued six days. He then experienced almost complete relief. But on the same day he was suddenly attacked with pain below and inside the left nipple, which was atrocious, and continued during the succeeding night. The next morning the pain continued so severe as to make the patient cry out. It was not increased by pressure, nor by coughing, nor by taking a full inspiration, nor by motion. The action of the heart was tumultuous, very much accelerated, irregular in force, and intermittent. The pulse was also intermittent, difficult to be felt, and sometimes insensible. The joints were entirely free from rheumatism. The chest sounded well on percussion, the respiratory murmur was puerile, there was no cough. The countenance was pale, contracted, and expressive of the most intense anxiety; the extremities were cold. Although pericarditis was recognized, the patient appeared too feeble to be bled again from the arm, but thirty leeches were applied to the precordial region, and sinapisms to the extremities.

No improvement followed the treatment. The dyspnœa became more and more marked, and the patient died the following night, twenty-seven hours after the attack.

On post-mortem examination, the whole internal surface of the pericardium was lined by a white, soft, and areolar membrane, and contained about an ounce of greenish serous fluid. The pericardium itself was of a bright-red color. The muscular substance of the heart, and the cavities, were healthy. The lungs were congested with a bloody serum. The other organs were all healthy.

Professor Andral, from whose Clinique Médicale this case is abridged, regarded the effusion as purulent in its character, that is, the liquid effusion. It is a striking illustration of metastasis from the joints to the heart, and that, too, after repeated venesections. Thus, tending to confirm the doctrine maintained by Fordyce and by others, that free depletion in acute articular rheumatism, is apt to be followed by a metastasis to the heart. Other observers, however, have not confirmed this opinion, and the result of general experience seems to be, that this metastasis is merely an accidental circumstance.

There were no physical signs observed in this case. It is probable that none existed. But it should be recollected that the case was observed at a time when the physical diagnosis of pericarditis was much less understood than at present. But the rational symptoms were so well marked that no one could hesitate in the diagnosis. It is singular, however, that the pain was not aggravated by pressure and by motion, and that none of the tendency to fainting was observed, which is so frequent in this severe form of the disease.

The *rational symptoms* of pericarditis are few in number, and frequently indistinct, or even absent. This is one reason why the disease is so frequently latent. Pain in the precordial region may be present, but if so, it is not always severe. In some cases, however, the pain and distress is atrocious, drawing attention at once to the heart. In other cases it is trifling, or only induced by means directly employed to excite it. Sometimes it is felt only in the epigastrium, or even more remotely from the heart. It is increased by a full inspiration, by percussion, by

pressing upward towards the heart from the left hypochondrium, but more especially, by whatever suddenly excites the action of the heart, as motion, or mental excitement. This pain sometimes assumes the character of distress, and is accompanied by a sense of constriction and anxiety. If the attack coexist with severe articular rheumatism, the pain, although existing, may not excite complaint, being absorbed, as it were, by the pain in the articulations.

Another rational symptom is palpitation. It is quite characteristic in some of these cases. You will find the patient complaining much of this symptom, and yet when the hand is applied to the precordial region, the action of the heart may not be felt at all. The reason of this is apparent—it is caused by the effusion of serum. Palpitation, like pain, is chiefly excited by whatever suddenly increases the action of the heart.

The *physical signs* of this disease are far more striking than the rational symptoms. They depend, for their existence, upon the two effusions of serum and of lymph. It is easy to understand, at once, what must be the effect of a copious effusion of serum into the pericardium. The heart is forced backward towards the spine, so that its impulse against the ribs becomes feeble, or ceases altogether, the sounds of the heart become indistinct and distant, while the dulness over the precordial region is much increased, the respiratory murmur is absent, and even a projection of the precordial region may be observed. But this is not all. The effusion of lymph follows, and the friction of the opposite surfaces roughened by this deposit, generate a friction sound, which is, of course, most distinct when the serous effusion is moderate in quantity, but is sometimes heard when the effusion of serum is very considerable. The existence of ten or twelve ounces of serum in the pericardial sac will not necessarily prevent you from hearing it. This friction sound is sometimes accompanied by a distinct thrill.

The influence of the two effusions in producing the physical signs is so simple, that you might almost *a priori* determine what they must be. To state these signs in connection with the appearances found in fatal cases might almost seem sufficient,

but there are some of them which demand a more extended consideration.

The dulness on percussion is usually very much increased, both in extent and in degree. It may extend upward to the left clavicle, to the right of the sternum, as far as the right nipple, and to the outside of the left nipple. In most cases, however, it is much less extensive. If the serous effusion on which it principally depends be small in amount, it accumulates behind the heart, and the dulness on percussion will be trifling. Or if the lungs are voluminous, so as naturally to cover well the precordial space, this will diminish the dulness. Generally, however, decided dulness extends upward to the second or third rib, across the sternum to the right, and to the nipple on the left side. This may be regarded as a fair development of dulness. Besides, the intensity of the dulness over the centre of the precordial region is much increased.

The tendency to dilatation of the parietes of the chest over the precordial region is equally, but not so decidedly or so frequently, an indication of the amount of serous effusion. It indicates a considerable effusion, and must be carefully distinguished from the frequent bulging of the ribs in this region, dependent upon their malformation. Unless accompanied by an equal prominence of the intercostal spaces, by dulness on percussion, and the other physical signs of copious effusion, it possesses no value.

When the acute inflammations of the heart, pericarditis and endocarditis, first attract ed the attenton of auscultators, and for a long time afterwards—indeed, the difficulty is not yet entirely removed—it was very difficult to distinguish the morbid sounds generated by these two diseases. The true basis of diagnosis may rest upon the ability to distinguish a friction sound from a blowing sound. But can this always be done? If I give you my own experience on this point, I must say that it cannot. A blowing sound gives the sensation of the bellows blast, a friction sound gives the sensation of the rubbing of two rough surfaces together. But the latter may possess a softness that makes it very like a bellows murmur, and the former often possesses a harshness that sounds much like a rubbing sound, and vice versa.

Thus, while the character of the sound, taken alone, will not always guide you to its true nature, yet it frequently will do even this. The ear at once distinguishes the blowing or the rubbing sound. Fortunately there are other circumstances to guide you, to which I will only allude at this time, as they can only be properly appreciated when you compare them with those which occur in endocarditis. The friction sound is usually double, although it may accompany either the contraction or the dilatation of the heart alone; it is diffused over the heart, or, when heard more distinctly over a circumscribed portion, as the apex or the base, it is apt to change its position from day to day. It has been stated by some good auscultators, that the sounds are more superficial than in valvular disease; also, that they may be modified, or even made to cease, by changing the position of the body, as from the left to the right side, but I have never been able to satisfy myself that this is so. The thrill which sometimes attends these cases is also usually extended, and liable to vary in intensity in different points, from day to day, during the progress of the case.*

I have already remarked, that a considerable effusion of serum will not prevent the friction sound from being heard. It is also probable that at the very commencement of the disease, when the serous surfaces are dry and sticky, that a soft friction sound is generated. Such cases are so rarely seen in practice, that the fact can hardly be established. It is safer to attribute the friction sound, when you hear it at any period of the disease, to the presence of effused lymph. Now, as the lymph varies very much in different cases, in quantity, in consistence, and in the degree of roughness, the friction sound must equally vary. If the membranes are limited in extent, the friction sound will also be limited. If the false membranes are at the base of the heart, you will hear the sound over the base; if at the apex, you hear the sound over the apex; while if they exist only posteriorly, you may hear the sound most distinctly between the scapula and the spine. Again, if the false membranes are soft and smooth,

* The existence of a thrill in pericarditis is rare. In 13 cases in which the rubbing sound was heard, it existed but twice.

the sound will be proportionately soft, approaching the blowing sound. If they be rough and firm, the sound will become harsh and grating, or creaking in its character. The force with which the heart is acting will also modify the character of the sound, increasing its harshness when it is violent. I have also stated that the sound is usually double, but this is not always the case. When single, I have commonly found it masking the first or systolic sound of the heart, but in some cases it masks only the second—the diastolic sound. Dr. Taylor, in his excellent Report of cases of pericarditis in the London Lancet, states that it is usually, when double, more strongly marked with the diastole than with the systole of the ventricles.*

The friction sound may accompany the disease from a very early period of its existence until near its termination—that is, from the time when the effusion of lymph takes place until adhesions have formed, or until the lymph has been absorbed. It is impossible to foresee how long this may be. In the examination of many cases, the shortest duration was three days, the longest was twenty-eight days. This sound frequently ceases for a time, to reappear during the progress of the case. The effusion of serum may become so considerable, and separate the opposing surfaces so widely, that all friction of the false membranes is prevented. A feeble action of the heart will also favor this cessation of the friction sound. But when the serum is partially absorbed, the opposing surfaces come again in contact, and the friction sound returns. If you will reflect a moment upon the attending circumstances of the case, you can hardly mistake this temporary cessation of the morbid sound for that caused by adhesion, or by absorption of the effused lymph. In one case the signs of serous effusion, viz., precordial dulness and dilatation, the distance of the sounds of the heart would exist in a marked degree; while in the latter case they would be entirely absent.

The friction sound is not heard in all cases of pericarditis,

* In 18 cases of rheumatic pericarditis, the friction sound was double in 17 cases. In six cases, the diastolic sound was the loudest, but in several cases in which the friction sound was forming, it was single and systolic. In six cases, the friction sound was loudest at the base of the heart; in three cases, it was loudest at the apex.

even when they are carefully watched. In eight cases, reported by Hache, it was wanting in five cases. This is owing to the rapid and abundant effusion of serum, especially in the subacute form of the disease. False membranes are so generally found in this disease, that you can hardly attribute the absence of the friction sound to their non-existence. You may better attribute this, especially when the sound is not heard after a considerable portion of the serous effusion is absorbed, to the rapid formation of adhesions; or, perhaps, in some cases, to the fortunate absorption of the effused lymph.

It has been remarked, that the friction sound is more frequently heard in cases of rheumatic pericarditis than in those cases in which the disease is primitive, and this has been supposed to be owing to the comparative smallness of the liquid effusion in the former class of cases. But, probably, the vigorous action of the heart in these cases has some influence on this result. In cases connected with Bright's disease, and in many cases of primitive pericarditis, the excitement of the circulation and the force of the heart's action is much less than in the cases which complicate acute articular rheumatism.*

You might be led to hope that resolution with absorption of the effused lymph, was the natural termination of acute pericarditis, especially as this is true of the effused serum. But this, I fear, is not the usual termination. There are remedies that possess an undoubted power in producing the absorption of effused lymph, yet you will frequently fail in effecting this complete result. Unfortunately, there are no characteristic signs which will enable you to determine when the disease has terminated by resolution and when by adhesion. In both cases the friction sound ceases, and the heart may apparently return to its natural condition. It has been contended that adhesion of the pericardium is indicated by a jogging action of the heart.

* In an analysis of eighteen cases of rheumatic pericarditis, the friction sound was heard in every case. While in nine other cases not rheumatic, five of which were complicated with Bright's disease, the friction sound was distinctly noticed in but three of the cases. In the eight cases reported by Hache, only two of which were rheumatic, the friction sound was heard only three times. But whether the friction sound was heard particularly in the rheumatic cases is not stated.

Again, it has been stated that an undulating movement has been perceived over the precordial region. But in either case the fact is but occasional, and even then not diagnostic, at least in the latter instance. In a few cases a depression over the precordial region has been observed. This condition, if it has been preceded by the symptoms and signs of pericarditis, is much more conclusive of the formation of adhesions.

The constitutional symptoms of acute pericarditis are, for the most part, those of other acute inflammations. The attack is frequently ushered in by a chill, especially when the disease is primitive. This is followed by febrile reaction, according to the violence of the disease and the individual susceptibility, and by other symptoms, some of which are so intimately associated with the disease as to deserve a particular notice. The functions of the lungs, organs so intimately associated with the heart, are always affected: thus you will notice dyspnœa, and frequently cough. Both these symptoms are often associated with actual disease of the lungs, as pleurisy, pneumonia, or bronchitis. But they may exist independently of those conditions. Whatever increases the disturbance of the circulation in this disease, aggravates the dyspnœa; and the dry and trifling cough often appears to be only the effect of a secondary irritation, increasing in severity if pulmonary inflammation supervenes.

The nervous system is also sometimes much affected in this disease. Among the most striking symptoms, in aggravated cases, is the sense of anxiety which stamps itself upon the features, a disposition to jactitation and uneasiness, a tendency to syncope, a small and irregular pulse, and sometimes even delirium, convulsions, or coma. The occurrence of these symptoms may be referred, in part, to a susceptible nervous organization; but, as a general rule, they are found to occur either in cases in which the inflammation of the pericardium is of a very high grade, or when some secondary inflammation, especially that of the lungs or pleura, is known to coexist. Nothing, however, is more common in cases of pericarditis of ordinary severity, than to find the patient lying quietly in bed, with the countenance calm, the pulse regular, the febrile excite-

ment moderate, and with no complaint of distress, unless the action of the heart is excited. In other cases, in which the pericarditis is secondary, and frequently latent, the constitutional symptoms may be in nowise different in degree, or in character, from those belonging to the original disease.

When, however, pericarditis is associated with pleurisy, or with pneumonia, although the symptoms of the original disease predominate, yet there is usually something in the case to draw the attention of the careful observer to the heart. Thus, the tendency to fainting, great dyspnœa, the irregular pulse and other disturbances of the nervous system, such as occur in the more severe and fatal form of pericarditis, may all be induced by a coexistent inflammation of the lungs or the pleura, the pericarditis itself possessing no unusual severity—complications which add also very much to the fatality of the disease, as indeed might be expected.

Pericarditis is also frequently associated with acute articular rheumatism, and in this case the complication is marked by a different character in the symptoms. Instead of presenting an aggravation of the symptoms belonging to the organs in the chest; of the dyspnœa, the palpitation, the pulse, as well as the tendency to fainting, the disease is frequently entirely latent. The pain in the joints, usually so distressing, seems to absorb the sensibilities of the system, and you may be only led to the detection of the pericarditis by the recollection of its frequent occurrence in connection with rheumatism, and by the study of the physical signs.

Chronic pericarditis is not only a rare form of disease, but its symptoms may readily be confounded with those of enlargement of the heart. And I would remark, that you cannot usually regard the adhesions that form in consequence of acute pericarditis, as constituting a chronic form of the disease. Generally, when adhesions have taken place, the inflammation is at an end, and this may occur as early as three or four weeks. In some cases, however, the false membranes, under the influence perhaps of a low degree of inflammation, become fibrous, cartilage-like, or even bone-like, and produce symptoms both rational and constitutional, as well as physical signs, which cannot, so far as I know, be dis-

tinguished from those of enlargement of the heart. Evidences of an obstructed circulation ensue, as well as signs of enlargement of the heart, caused by the thickened and altered false membranes that invest it, and that even when the muscular substance of the heart is atrophied by their pressure. Cases in which purulent matter continues in the pericardium as the consequence of acute inflammation, are very rare, as such cases, indicating either a very high grade of inflammation, or a vitiated state of constitution, usually terminate fatally at an early period. Occasionally, however, cases occur which, beginning insidiously, are followed by an effusion of serum, with very little lymph, or by a sero-purulent fluid, and continue much beyond the ordinary period, constituting an essentially chronic form of this disease. In these cases, in addition to the physical signs of liquid effusion into the pericardial sac, you may notice a slight degree of febrile reaction, especially in the evening. With this general condition, is associated dyspnœa, uneasiness, or sense of weight in the precordial region, with slight puffiness in the face and around the ankles.

The diagnosis of acute pericarditis is not always easy, even when your attention has been drawn to the examination of the heart. It is sometimes difficult to distinguish it from endocarditis, especially when only a small effusion of serum exists in the pericardial sac. But I shall not attempt to point out the differences between these two affections at the present time. This can be much better done when I have placed before you the leading symptoms of endocarditis. It is important to remember that pericarditis is not very unfrequently a latent affection, especially when it occurs as a complication of rheumatism, of Bright's disease, of pleurisy, of pneumonia, or in the course of fevers. In those diseases, therefore, in which pericarditis is most apt to occur, the heart should be carefully watched. In acute articular rheumatism in particular, it is a standing rule in this Hospital that every patient with the disease shall have his heart examined every day.

Chronic pericarditis may be confounded with enlargement of the heart. In both diseases there is increased dulness in the precordial region. In both, there may be a prominence of the

chest in the same region; in both, there may be a feeble action of the heart. But here the resemblance ceases. Pericarditis drives the heart away from the precordial region, enlargement of the heart brings it nearer to the surface. Pericarditis causes the apex of the heart to cease to beat against the anterior parietes of the chest, or leaves it in its natural position between the fifth and the sixth ribs. Enlargement of the heart carries the apex to the left of its natural position. But these, and other points of difference, will be better understood when you have studied the physical signs of enlargement of the heart.

LECTURE XXVII.

ENDOCARDITIS.

Physical signs and rational symptoms.—Causes.—Treatment of pericarditis and of endocarditis.—Venesection, leeching, mercury, digitalis, diuretics, tonics.

Before I speak of the treatment of pericarditis, I wish to call your attention to another form of acute inflammation of the heart —that of the lining membrane, or endocarditis. My reason for so doing will be obvious, when you understand that not only the principles that regulate the treatment of the two diseases are identical, but that, in a majority of instances, they recognize the same cause, and that they frequently coexist. I shall confine myself, however, to the acute stage of the disease, reserving what I may have to say on the more remote consequences of this inflammation to my lecture on valvular disease.

In acute endocarditis, you will find after death redness, thickening, and perhaps softening of the lining membrane of the heart, and especially of that portion forming the valves. Frequently the effects of the inflammation are confined to the valves, and are most frequently seen in those of the left side of the heart.* Sometimes the injection and softening belong more par-

* In nine cases, in which the lining membrane of the heart was evidently in

ticularly to the subjacent cellular tissue; so that the lining membrane can be more readily peeled off than is natural. With this condition of the lining membrane you may find an effusion of lymph, either in the form of patches, or of granulations, especially upon the valves, and, in some rare cases, a secretion of pus, and possibly ulceration, and even gangrene.

Fibrinous concretions are often found in the cavities of the heart in acute endocarditis, of a pale rose or yellowish tint, firm, elastic, adhering to the lining membrane, and twisted among the tendinous chords. What is their nature? They have been supposed to be owing to inflammation, or at least to a tendency to coagulation in the blood as it is passing through the heart, and in contact with an inflamed tissue. But you will find the same concretions, or polypi, as they are called, in many cases of enlargement of the heart, in which no endocarditis exists, and in which there is a simple stasis of the blood from obstruction to the circulation through the heart. Finally, you will notice the same tendency in almost all cases, even when the heart is healthy, in which the agony has been prolonged and the heart has been congested during the last hours of life. It appears to me, therefore, that the stasis of the blood is the chief cause of these polypi, and that they are distinct from the effusion of lymph, which may exist either with or without them, although inflammation of the lining membrane of the heart may favor their formation.

The great cause of endocarditis is acute articular rheumatism; and the remarks that I made in relation to this cause when speaking of pericarditis, apply precisely to endocarditis. The researches, however, of Dr. Taylor, already quoted in relation to the frequent association of Bright's disease with pericarditis, apply equally to endocarditis. Thus, this observer found acute endocarditis in about eight per cent. of his cases of Bright's disease. In fifty post-mortem examinations in which Bright's disease distinctly existed, he found acute endocarditis in one of twelve cases, and the consequences of the disease in nearly one-half of

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flamed, the left side of the heart was affected in all of them, and four times without a similar affection of the right side.

the cases. It is possible that phlebitis may sometimes lead to the production of endocarditis by the extension of the inflammation to the lining membrane of the heart, or by metastasis; but such cases are very rare.

The rational and the constitutional symptoms of endocarditis resemble so nearly those of pericarditis, that by relying upon them alone you could not be certain as to the diagnosis. In a severe case of endocarditis, there is pain or uneasiness in the precordia, and palpitation. In cases in which there is much obstruction to the circulation from swelling of the valves, or from the formation of polypi, dyspnœa is present; in other cases it may not be noticed. When much obstruction to the circulation exists, the same severe symptoms noticed in the worst cases of pericarditis are present, viz.: a tendency to syncope, a weak and irregular pulse, delirium, convulsions, and even coma. In all such cases, you must carefully examine and discover if the signs of pericarditis, or of pleurisy, or of pneumonia are not also present. There is in this acute form of the disease more or less constitutional fever; and, unless in very severe cases, the pulse is regular, although accelerated.

If you examine the region of the heart, you will commonly find the impulse regular, superficial, and sometimes strong and tumultuous; sometimes stronger than the pulsation at the wrist. You will find the apex in its natural position, and the dulness over the precordial region not materially increased. Cases, however, have been recorded, in which the precordial dulness has been found somewhat increased, owing, perhaps, to a distension of the cavities of the heart from congestion. It has also been observed in such cases, and not unfrequently, that the heart becomes irregular in its action, and the impulse feeble, probably from the same cause. It is in connection with this condition of the heart that intense dyspnœa and anxiety, as well as other distressing nervous symptoms, are noticed to exist.

In listening to the sounds of the heart, they may be simply muffled and obscure. But few cases, however, present so indistinct a change. Almost always—the exceptions are very few—you will hear the blowing sound; and this occurring suddenly, and existing distinctly, is, after all, the characteristic sign of

acute endocarditis. It varies in its character, according to the degree of roughness, or obstruction, that may exist, and according to the force of the heart's action. It varies in its seat, according to the valve affected. These are points, however—delicate points in diagnosis—which I shall fully discuss when speaking of valvular disease in another lecture. It is sufficient to remember for the present, that a blowing sound, suddenly developed over the heart, is the sign of acute endocarditis, and that it is to be carefully distinguished from the friction sound of acute pericarditis. I must, however, mark at this time one important exception. In cases of anemia, from loss of blood, a blowing sound, possessing certain characters, may be suddenly developed in the precordial region, which has no connection with endocarditis. The characteristics of this inorganic blowing sound will also be fully described in my lecture on valvular disease. I will only remark now, that the mistaking this sound for that belonging to endocarditis is a capital mistake, yet one that, I believe, has often been made. Thus, those who are in the habit of bleeding profusely in acute rheumatism, may develop this inorganic murmur, and falsely attribute its existence to endocarditis.

But all cases of endocarditis do not present the severe or well-marked symptoms I have just described. By far the larger number of cases are much milder in their nature, and many of them would be entirely latent were it not for the development of the blowing sound. There may be no marked increase in the action of the heart, or this, if it exists, may readily be referred to the coexisting rheumatism. The patient may experience slight pain in the precordia. Or no precordial pain may exist, or dulness on percussion, but only a simple blowing murmur. If there is no reason to suspect that anemia exists to create a blowing sound (which I may now observe always attends the first sound of the heart, and has its seat over the aortic valves), you can safely refer it to a slight endocarditis, probably confined to the valves. This valvular murmur may be heard day after day; it frequently, I think generally, continues after the acute disease with which it is connected has subsided; and finally, the morbid changes that induce it may lay the

foundation for incurable enlargement of the heart. A majority of the cases of comparatively latent endocarditis, occur in connection with acute articular rheumatism.

It will now be easy, I think, to distinguish a case of acute endocarditis, even when it assumes the mild and insidious character it often does assume, from acute pericarditis. I have already alluded to this subject when speaking of pericarditis, and will repeat now what I said then, that the rational and constitutional symptoms will not often aid you materially in the diagnosis; they are so much alike in most cases. But even in the more acute form of endocarditis, the action of the heart continues superficial; there is no effusion to push it backward as in pericarditis; there is not much, if any, increased dulness on percussion; there is a blowing murmur instead of a friction sound, confined to the region of the valves, or to the apex, instead of being diffused over the heart. This sound is usually fixed, instead of being movable from day to day—often heard only at the apex; and finally, it often continues long after the other signs of inflammatory action have subsided, lasting for years—for the remainder of life.

I cannot help alluding still further to the characters of the two characteristic morbid sounds heard in cases of acute pericarditis and of endocarditis—the friction sound and the blowing sound: for I am well aware, that no subject connected with the science of auscultation has puzzled its followers more than this. For a long time no accurate distinction was made in the character of these sounds—they were constantly confounded with each other. After a time, however, the true distinction between a blowing sound and a friction sound was established. The ear became accustomed to discriminate them by the fundamental character of each—the kind of sound. Other circumstances, already alluded to, were noticed in connection with each. The superficial, diffused, and movable nature of the friction sound, as well as its comparatively short duration, and its being usually double, were carefully noted. While the blowing sound, by its greater concentration and fixedness, its tendency to pass along the course of the great vessels, or to exist

at the apex, by its being usually single,* and continuing for a long period, became impressed with marks, which, although not perfectly characteristic, are yet often of great value in doubtful cases. If you could readily distinguish a blowing sound from a friction sound in all cases, that would be enough for the diagnosis. But you cannot. A friction sound may hardly be distinguished from a soft blowing sound, and both may become equally harsh and grating.

I will relate a case as an illustration of the symptoms and progress of acute rheumatic endocarditis which terminated fatally.

A seamstress, aged 27 years, usually in the enjoyment of good health, was attacked with severe acute articular rheumatism, for which she was bled four times, in three days, to the extent of 84 ounces, commencing on the 9th day after the attack, and by full doses of opium at night, but without marked benefit. On the 13th day, some tenderness on percussion was observed in the precordial region, and a slight blowing sound was noticed with the systole of the heart. There was no dulness on percussion, and the respiratory murmur was distinctly heard over the heart. Repeated examination before this time, revealed nothing abnormal in the action of the heart or in the lungs. The next day, the blowing sound was more distinct, and more extended. It was heard over the whole precordial region. About the nipple, it was rather rough and rasping in its character. There was no dilatation over the precordial region, but the tenderness on percussion continued. The pulse was 100 in a minute, large, and regular. There was no improvement in the condition of the joints. The patient was bled again, to the extent of 18 ounces. In the evening, the rheumatic inflammation of the joints was much worse, but the blowing sound was less distinct, and its rasping character was gone.

* Dr. Budd states, that in twenty-three cases of rheumatic endocarditis, observed with much attention, the blowing sound was diastolic in only one case; in all the others, the systolic sound was exclusively altered. In all but three cases, the sound was much louder over the left than over the right cavities of the heart.

During the five following days, the patient was bled twice, moderately, to the extent of 26 ounces, and used opium in large doses, but still without marked relief. The condition of the heart continued unaltered, except that, on one occasion, and for a short time, a slight friction sound was heard, two inches to the right of the sternum. The respiratory murmur continued natural, and with from 20 to 30 inspirations a minute. The pulse was soft, and varied from 100 to 116 in a minute.

On the 18th day, the patient seemed quite prostrated, and the tongue was rather dry. The rheumatic inflammation of the joints remained stationary. The pain in the precordial region had increased, and the blowing sound continued; but there was no dulness or dilatation over this region, and the respiratory murmur could be heard there. There was no syncope, œdema, or delirium. The pulse was regular, of good strength, 116 in a minute; respiration was 30 in a minute. Twenty leeches were applied to the precordia. In the evening, the pulse had fallen to 100, and was slightly irregular; the respiration had increased to 40 in a minute. The blowing sound could hardly be heard. The patient was much prostrated.

During the following week, the patient seemed to improve; the pulse became regular, the pain in the precordial region subsided, although, at one time, the inflammation of the joints increased, and the patient was bled again, 13 ounces. The blood drawn at different periods was always buffed, but the proportion of serum was gradually increased. The inflammation of the joints diminished.

On the 27th day, the respiratory murmur was found to be feeble at the base of the right lung, with slight dulness on percussion, but without pain or chills. Six days before, a cough, with sibilant rhonchus on the right side, was noticed. The pulse was 106 in a minute, and regular. The sounds of the heart were natural.

The following day, evidences of pneumonia of the right lung existed in combination with the pleuritic effusion, indicated by increased dulness, bronchial respiration over the inferior two-thirds of the right lung, with viscid expectoration and egophony. The respiration was 44; the pulse 108 in a minute, and feeble.

There was no lividity or œdema, but agitation. In the night, the patient died, 19 days after the heart became affected.

The brain presented nothing remarkable. There was a serous effusion, amounting to a pint, in the right pleural sac. The lower lobe of the right lung was dense and unaerated, but presented no traces of pneumonia.

About two spoonsful of clear serum was found in the pericardium. There was a small patch of thin and recent false membrane, an inch and a half square, on the posterior surface of the heart. The organ itself was larger than natural, its muscular structure red, and of good consistence. The cavities of the ventricles were sensibly dilated, and all the cavities contained black clots of blood, which did not adhere to the walls. The left auriculo-ventricular orifice was surrounded by vegetations of the size of a pin's head, united with each other or separate, grayish and tolerably firm. The chordæ tendineæ were enveloped in a grayish, granular, friable matter, which adhered to them slightly. On a portion of the mitral valve this matter formed a mass, eight lines in thickness. Another mass, 10 lines thick, projected into the cavity of the left ventricle, near the origin of the aorta—to the lining membrane of which ventricle it adhered with some firmness—and when scraped off, exposed the lining membrane thickened, opake, and of a grayish color. The above mass was soft, grayish, granulated upon its surface, spongy internally, and somewhat elastic. Along the free edge of the aortic valves the same vegetations existed, also in the depression between the valves, and resembling syphilitic vegetations in appearance. The same vegetations, but much less developed, existed around the right auriculo-ventricular orifice. The abdominal viscera contained nothing worthy of remark.

This case, reported by Grisolle, is worthy of attention in many respects. The anatomical appearances are well described, presenting an endocarditis after many of the more acute morbid appearances had subsided. The patient died on the 19th day. Had death occurred at an earlier period, the lining membrane of the valves would probably have been found red and swollen, but this condition had passed away, leaving opacity, thickening, and a grayish tint. Still the lymph effused and existing as ve-

getations and in masses, was not yet organized, and it does not appear that any contraction or deformity of the valves existed, beyond what would be caused by the simple mass of new matter deposited; there was no shrivelling, no rigidity noticed. The cavities of the heart were dilated, while the thickness of the walls was, perhaps, rather increased.

The only rational sign of endocarditis was tenderness on percussion, and the only physical sign was the blowing sound heard with the systole of the heart. No attempt seems to have been made to diagnosticate the particular seat or form of valvular disease. The practical fact of endocarditis was, however, recognized. I may remark, that the attack came on after repeated bleedings, and that neither it, nor the accompanying rheumatic inflammation of the joints, were much relieved by it. It is to be regretted, I think, that the patient was not treated by mercurials, which have been found so beneficial in such cases, both by English and by American practitioners, but to which the French have a very strong prejudice. The case was aggravated by a serous effusion into the right pleural cavity, preceded for several days by the evidences of bronchitis of the corresponding lung. It is worthy of remark, that a bronchial respiration was developed as the simple result of liquid effusion compressing the lung—a fact I have noticed in other cases, but generally unconnected with adhesions, or with some cause that obstructs the pulmonary tissue, as pneumonia or tubercles; but nothing of this kind existed in this case.

Finally, I may remark, that the sign of endocarditis ceased some days before death. This cannot be accounted for by the condition of the orifices of the heart, which probably remained unchanged. The true explanation, I think, is the increased feebleness of the heart's action, which prevented the development of a blowing sound, even when a good deal of physical obstruction existed. It will also be remembered that it became more feeble immediately after the patient had been bled.

Cases of subacute endocarditis are constantly occurring in my Hospital patients affected with acute inflammatory rheumatism. They are often latent, or revealed only by trifling pain and palpitation. They are indicated chiefly by a blowing sound, and

this blowing sound is usually systolic and heard at the apex, indicating regurgitation at the orifice of the mitral valve. My observation has led me to believe that in a large proportion of the cases of rheumatic endocarditis, the mitral valve is the principal seat of the disease. It also happens, I think, in a large majority of cases, that this blowing sound continues after the rheumatism has disappeared, and that the foundations of incurable valvular disease and of enlargement of the heart are already laid, which may develop themselves by distinct symptoms—only perhaps after an interval of years.

A lady of rather delicate constitution, twenty-eight years of age, experienced two attacks of acute articular rheumatism, in neither of which was any affection of the heart suspected. Two or three years after the last rheumatic attack, she began to experience the symptoms of heart disease, dyspnœa, palpitation, and when I first saw her, three years after these symptoms had ensued, I found a slight enlargement of the heart with disease of the mitral valve. She continued under my care for six or seven years, during which time the disease made no sensible progress, until at length, after a long interval of unusual alleviation of her heart symptoms, she died of an acute attack. The heart was but little enlarged, but the mitral orifice was reduced, by former inflammation, to a mere chink which would not admit the end of my little finger.

Such cases, indicating a slow progress in the changes produced by rheumatic inflammation of the valves, are by no means rare. I will mention another case which was apparently cured, but in which the physical signs remain, and will probably at some future period lead to the existence of serious symptoms.

A little boy, aged six years, of good constitution, and generally enjoying good health, had been attacked about three weeks before I saw him with acute articular rheumatism, which was moderate in degree, and soon yielded to treatment. A few days before I saw him, the attending physician was recalled, and could detect nothing abnormal but increased and very rapid action of the heart. When I saw him, I discovered a double pneumonia, most marked in the right lung. The pulse was accelerated, but there was little fever. The respiration was also

accelerated somewhat, and there was some cough, but no pain in the chest. Indeed, the constitutional and rational symptoms were quite moderate in degree. The impulse of the heart was increased, and a blowing, systolic sound was noticed, chiefly towards the apex, which struck between the fifth and sixth ribs, a little to the left of the nipple.

The treatment by mercurials was commenced at once, and the gums slowly became affected. By this treatment, the pneumonia rapidly subsided, and the general condition of the child improved. He enjoyed his food, he slept well, he gained strength, and was free from cough and fever. But the blowing sound over the heart continued, at times even with a thrill. The apex continued to strike to the left of the nipple, and there was considerable dulness, and perhaps some dilatation over the precordial region. The pulse was 100, soft and regular; respiration 22; countenance rather pale.

The mercurial treatment was continued moderately, with slight salivation, for two months, during which time the general health improved, and no symptoms of cardiac disturbance existed; but the physical signs above mentioned continued. The impulse of the heart was quite natural.

I examined this child two years and seven months after the attack. He has continued to enjoy excellent health, and has not suffered from rheumatism or from the slightest symptom of cardiac disease. He plays and runs about with other children, without dyspnœa, palpitation, or pain. But on examination of the precordial region, the same physical signs exist, precisely as they did two years and a half ago. There is still some dulness and a little dilatation over the precordial region; the apex strikes rather too far to the left side, and the blowing, systolic sound towards the apex is still heard. The impulse of the heart is moderate.

The *treatment* of acute pericarditis presents the same indications as other serous inflammations. Active antiphlogistic remedies are clearly indicated in those cases which are violent in their attack, and which occur in young and vigorous constitutions, especially when the disease is at an early stage. The same indications present themselves when the disease, less vio-

lent in itself, is complicated with pleurisy or pneumonia; also in cases of rheumatism, when the constitutional excitement is strongly marked. In such cases, your most potent means of relief are, abundant and repeated venesection, followed by the application of cups and leeches to the precordial region. After depletion has been carried as far as the circumstances of the case seem to warrant—that is, after the force, hardness, and frequency of the pulse are reduced—your chief reliance must be on the free use of calomel combined with Dover's powder, or with some form of opium. The constitutional effects of mercury should be induced as early as possible. The patient should also be kept as quiet as possible, and on the lowest diet. The bowels should be kept moderately open; but active purging, which is apt to distress the patient by the movements it causes, should be carefully avoided. It must be a very bad case that does not show decided symptoms of amendment when the antiphlogistic treatment has been well carried out, and the gums become affected by the mercurial action. The case is, then, usually under your control, and you may watch the gradual absorption of the serum, then the cessation of the friction sounds, either from absorption of the lymph or the formation of adhesions. The subsidence of the constitutional symptoms, and the more or less rapid return of the heart to an apparent, rather than real, normal condition, may be predicted; for adhesions of the pericardium probably continue.

The great object you should keep in view after the violence of the disease is broken, is the absorption of the effused lymph. As I have already remarked, you can never be certain that this has really happened; for the cessation of the friction sounds is quite as likely to occur from the formation of adhesions as from the absorption of the effused lymph. Still, you may hope to have effected this desirable change when the action of the heart rapidly and completely returns to its natural condition; when its action becomes quiet and regular, without morbid sounds, and the precordial region is free from every kind of uneasiness. I am inclined to believe that an uneasy feeling in the part affected is often a guide to the existence of adhesions after serous inflammations. Sometimes it is a sensation of weakness, some-

times of obscure pain, not constant, but apt to recur, especially after fatigue, or any cause tending to depress the vital powers; and, I may add, a tendency to palpitation from slight causes. It has been observed in some cases that a jogging action exists in the heart; in other cases, an undulatory motion has been noticed over the precordia; and in some rare cases, but which is, probably, the best indication of adhesion when it exists—a depression over the same region. But these indications do not exist, in the present state of our knowledge, to enlighten us in the great majority of cases.

Your best chance in effecting the absorption of lymph, is in the use of mercury, rapidly introduced into the system, and continued to the point of affecting the gums. The influence of this agent in favoring the absorption of lymph is abundantly proved; and although you will see its influence more strongly marked in certain cases, as in iritis, yet you will commonly find such a decided change in the symptoms and physical signs of pericarditis, as soon as the gums become affected, as to leave no doubt that an equally favorable change has commenced. The object in these cases is, not to cause a violent, but a rapid mercurial action; a violent action would only aggravate the symptoms, by increasing the irritability of the patient, and, at the same time, the action of the heart. The indication is rather to induce moderate tenderness of the gums, and to continue the action for a long time. The cessation of the friction sounds is not a sufficient indication to stop the mercurial action, for adhesions may have formed, which are yet capable of being removed by the absorption of the lymph not yet perfectly organized. I consider it prudent to continue its action for some time after the friction sound has ceased, if there be no contra-indications in the case; and until you are assured, again and again, by careful examination that the action of the heart is quite natural. You must be careful not to confound the irritation which the long-continued use of mercury, even if every precaution is taken, sometimes produces, for the lingering remains of the disease itself. My own impression, however, is, that you will frequently fail, with all the care you can use, in entirely preventing adhesions from taking place, although you may diminish their extent.

A remedy which I believe to be useful in acute pericarditis is digitalis. It exerts, in many cases, a controlling influence over the action of the heart, in moderating its violence, and the rapidity of its movements. It tends to promote diuresis, and thus becomes revulsive. It is used elsewhere as a general antiphlogistic remedy more frequently than in this country, but it seems particularly appropriate to the disease before you. It may be given in combination with calomel and Dover's powder, but it had better be used separately, although it need not interfere with the mercurial treatment. When given by itself, you can regulate its dose, and watch its effects better than when in combination. It does not appear to me to act rapidly, therefore it should be frequently repeated, and the dose increased. But you know that it is sometimes cumulative in its action; that is, from not acting apparently at all, it may suddenly act violently, and with all the symptoms of poisoning. As a general rule, I do not think that digitalis acts decidedly, either by controlling the heart or by increasing the urine, until its characteristic effects upon the nervous system are somewhat apparent. When you find the patient beginning to complain of indistinct vision, of a change in the natural color of objects, of muscæ volitantes, then you may find the heart more quiet, and the urine more abundant. I know that it is the opinion of some observers, that digitalis may act upon the heart alone, controlling its action. I can but state the result of my own observation. Digitalis, however, does not agree with all patients. It sometimes irritates the stomach, and thus increases, instead of diminishing, the action of the heart.

Diuretic remedies of various names have been much recommended in serous effusion connected with acute inflammation, with the supposition, perhaps, that they may act as they do, in promoting the absorption of simple dropsical effusions. But the cases are, in fact, widely different. In inflammation, diuretics probably act by reducing the inflammation itself, although they do not often act, at all, until the inflammation has passed its active stage. You will hear, indeed, of wonderful effects from the action of diuretics in inflammation—of pus even being carried off bodily, as it were, from the serous cavities, and

passed with the urine. I have no experience in such cases. Generally, I believe, when fluids, the result of inflammation, are absorbed, it is a proof that the disease is subsiding, and that the powers of nature are resuming their healthy action. That diuretics, or purgatives, or other derivative measures, as blisters, may assist this action, and even act directly in some degree in promoting absorption, I cannot deny.

All cases of acute pericarditis are not so severe as to require a very active antiphlogistic treatment. I may say, that a large majority of simple cases, and even of rheumatic cases, do not require more than one moderate general bleeding, followed by cups or leeches. Many cases, indeed, are so mild, that general bleeding may be entirely dispensed with; but all equally indicate the use of mercury. I have seen cases of pericarditis recover without this remedy, but who could say that adhesions had not taken place, and that these might not have been prevented by its use?

Acute pericarditis, when complicated with pleurisy, or with pneumonia, does not indicate any alteration in the general plan of treatment, although they may modify its employment. A case of pleurisy requires the same depleting and mercurial treatment as pericarditis, and although you might prefer, in a simple case of pneumonia, the use of tartar-emetic to calomel, yet the latter is a powerful means of resolving pulmonic inflammation—besides, the pericarditis is really the most important of the two diseases, from its remote effects. Of course, the activity of your antiphlogistic remedies must be proportioned to the extent of the inflammation, to its stage, and to the vigor of the patient—points of general practice that belong to all inflammations; but usually, when it attacks two different organs at the same time, an energetic treatment is indicated.

It has been observed, that relapses sometimes occur, even when the system is under the full influence of mercury. Be careful, however, not to see in this fact the inutility of this powerful agent. No remedial agent can prevent relapses, especially in a disease of a rheumatic origin.

You will sometimes find cases of acute pericarditis benefited by a treatment directly opposed to the antiphlogistic. When

the disease occurs in delicate or enfeebled constitutions, when it has been too actively treated at the commencement, when it has coexisted with inflammation of other organs and a state of anemia or exhaustion has followed, the progress of the cure may cease from the want of reactive power in the system. The case, examined from day to day, presents no improvement in the rational symptoms, or in the physical signs. The patient grows more pale and feeble, a tendency to œdema and night-sweats ensues. You find that your antiphlogistic remedies rather aggravate than improve the condition of the patient. It is now time to change your plan of treatment, and to administer tonics with moderation, allowing, at the same time, a more nutritious diet. The iodide of potassium, or the iodide of iron, either separately or in combination, and united with the decoction of sarsaparilla, are, I think, the best remedies. At the same time sleep may be procured at night and irritation quieted by small doses of opium. Soup, solid animal food, and even a little wine, may be useful in such cases.

There are cases which may be benefited by a mixed treatment. Mercury may be given in combination with tonics. In such cases, the protiodide of mercury may be used advantageously with the iodide of potassium or of iron. This treatment is especially indicated in the treatment of acute cases, which have been neglected at an early period, and have never been brought under the mercurial influence. It is also indicated in the treatment of chronic pericarditis, when these remedies may be combined with diuretics, and other means calculated to promote the absorption of effused fluids. Blisters are, I think, particularly indicated in this form of the disease, especially if it is attended by pain in the precordial region. They are often used in the acute form of the disease, but they are most useful in neglected cases, or in the chronic form of the disease.

There is another point in the treatment of acute pericarditis. After the inflammation has been subdued, the heart may remain irritable—subject to occasional attacks of pain and of palpitation. In such cases, iron, in combination with sedatives, is highly useful.

The treatment of acute endocarditis rests upon precisely the

same basis as that of pericarditis. Your reliance must be upon the use of blood-letting, and especially upon mercury. Blood-letting answers a double indication in severe cases of endocarditis. It is not only an antiphlogistic remedy of great power, but, by diminishing the mass of the blood, and by altering its quality, it tends directly to relieve the oppression and distress arising from an obstructed circulation, and especially from the formation of coagula in the cavities of the heart. But you must remember, that in this disease, as in pericarditis, blood-letting may be abused, and followed by unpleasant consequences—by irritative reaction, and the other effects of anemia.

The employment of mercury in subduing inflammation of the valves, and in promoting the absorption of effused lymph, is as strongly indicated in endocarditis as in pericarditis.

A question arises most important to answer correctly. What is the precise value of mercury in the treatment of pericarditis and of endocarditis? In almost every case you will find a decided amelioration of the symptoms, and of the physical signs of the disease, as soon as the mercurial action is established. But do these good effects continue and result in a perfect cure? My own impression is, that you will very often fail in producing this ultimate result. No doubt you will appear to succeed more frequently in pericarditis than in endocarditis. The reason is this: the symptoms and signs of pericarditis frequently cease when adhesions have formed, hence the cure may be apparent, not real; and you have often no means of determining the question. While in endocarditis, the case is quite the reverse. The blowing sound continues after the acute symptoms have ceased and the lymph has become organized upon the valves, or when a permanent thickening and deformity of these organs has ensued. But, especially in rheumatic patients, you cannot always be sure that the blowing sound is not the result of a previous attack, which may never have been suspected at its commencement, and may have been followed by a long interval of apparent health. You cannot expect, in such cases, that mercury will do any good. I have not data sufficient to establish the fact, how long an interval may elapse after the occurrence of endocarditis before irremediable changes in the valves may ensue. But I

am certain, from observation, that this interval may be very short. Indeed, you cannot be too early in the administration of your remedial agents. A few days' delay in detecting the existence of the disease, even in those mild, and often latent cases, in which the disease is limited, perhaps, to a single valve, or even to a portion of it, may defeat all your efforts, and you will be compelled to leave your patient to the chances of subsequent enlargement of the heart. My own decided impression is, that the reason why we so frequently fail in our efforts is, because we do not commence the mercurial treatment early enough, or use it actively enough. Dr. Latham has presented us with the results of his experience on this subject. In ninety cases of pericarditis, or of endocarditis, only seventeen left the Hospital without presenting some evidences, however slight, that the disease was not entirely cured, and yet but three of the ninety died of the immediate effects of the disease. My own experience tends to cohfirm, entirely, these facts. The sufferer from acute articular rheumatism, therefore, can only find consolation in the fact, that his disease is not often immediately dangerous. Frequently complicated with an inflammatory affection of the heart, which it is very difficult to cure entirely, it lays the foundation, often after years of apparent health, of a gradual, but progressive enlargement of that organ.

Before concluding what I have to say to you in relation to the treatment of rheumatic endocarditis, I wish to repeat, perhaps more distinctly, what I have already stated. Experience has satisfied me that the only chance of removing that apparently slight inflammation of the valves of the heart, which occurs so frequently in rheumatic subjects, is to attack it very vigorously and promptly. If the patient is vigorous, and there is much constitutional excitement, bleed freely from the arm in the first instance, apply leeches freely, and after short intervals, to the precordia, and induce the constitutional effects of mercury as rapidly as possible. Do not trust entirely to its effects when taken by the mouth, but use also the mercurial inunction. If the constitutional symptoms are very moderate, leeching may be resorted to without venesection. But it should be free, and

frequently repeated. By these vigorous means I have much more frequently succeeded in putting a stop to the blowing sound than when I pursued the same treatment, but with less activity.

LECTURE XXVIII.

ENLARGEMENT OF THE HEART.

Different forms of the disease.—Causes.—Rational symptoms; palpitation; pain.—Angina pectoris.

I PURPOSE, in this lecture, to speak of Enlargement of the Heart; and I use the phrase in a general sense, including all cases in which the heart is found larger than is natural. Enlargement of the heart may present itself in a variety of forms. In the first place, it may be either general or partial; that is, it may exist in both ventricles and auricles, or it may have affected one or more of these divisions of the heart. Sometimes you will find the enlargement confined to one side of the heart, as to the left ventricle and auricle; sometimes it is the right side only that is affected. Again, the enlargement, in whatever part of the heart it may occur, may exist in a variety of ways. Thus, the muscular structure alone may be increased in thickness and firmness, without other change: this is simple hypertrophy of the heart. In other cases, you will find the cavities of the heart increased in size, while the muscular substance remains unaltered, or, perhaps, is thinner than is natural: this is dilatation of the heart. But, in a great majority of cases, you will find these two conditions united; that is, you will find the muscular substance thickened and the cavities dilated. This is the common form of enlargement of the heart that is met with in practice, extending, in most instances, to all the divisions of the heart, although sometimes limited either to one side of the heart, or even to a single division, as to an auricle or to a ventricle.

Most authors who have written upon the diseases of the heart seem to me to have been too systematic in their arrangement of this subject, and, strange as it may appear at first sight, have rather confused than simplified the matter. Thus, they treat in distinct chapters, of Hypertrophy of the heart, dwelling at length upon its symptoms, its diagnosis, and its treatment. In the same manner, they treat of Dilatation of the heart—forgetting that in a great majority of cases these two conditions are united, and that, of course, their history, diagnosis, and treatment are blended. Now, while I readily admit the existence of simple hypertrophy, as found in a certain number of cases after death—few, indeed, in comparison with the whole number of cases—yet I doubt very much whether there are any certain indications, either rational or physical, by which you can determine its existence during life. This is still more strikingly true of simple dilatation of the heart. This form of disease undoubtedly exists, but it is, in my experience, exceedingly rare. The same remarks also apply, I think, to the attempts that have been made to distinguish enlargement of the right side of the heart from that of the left side. I do not doubt that this may sometimes be done with a certain degree of success, but not always, and when it is done I do not know that it leads to any practical result. Let me impress upon you that the great object is to ascertain the simple existence of enlargement of the heart. This is all that you can do in very many cases, and it is the great practical fact to be ascertained in almost every case. I wish to be understood as leaving entirely out of the question the subject of valvular disease, which is quite another matter.

Authors have also described a form of enlargement of the heart, which they call *concentric hypertrophy*, in which the muscular substance is thickened, while the corresponding cavity is diminished in size. There is much reason, I think, to doubt whether this is a genuine form of enlargement of the heart. I have never met with an unequivocal case of it. I have almost invariably met with this condition in cases of sudden death from hemorrhage, in which the heart has been rapidly emptied of blood and allowed to contract upon itself. Thus the walls become apparently thickened, and the cavities sometimes re-

duced more than one-half in size. This appearance has been uniformly noticed by me in cases in which no symptoms of cardiac disease existed during life. Indeed, the experiments of Cruveilhier appear almost conclusive on this point. He first noticed this appearance of concentric hypertrophy in decapitated criminals, and afterwards in animals who had been rapidly bled to death. I have already alluded to the influence of the stasis of the blood in the cavities of the heart, especially in those of the right side, in producing the opposite condition—an apparent dilatation of that organ. This is particularly true in cases of disease of the heart, in which the agony is often extremely prolonged, and in which organic impediments to the circulation cause an unusual accumulation of blood in the heart. It will frequently happen, when you first remove a diseased heart from the chest, that its size appears enormous, and that after removing the blood it contracts upon itself, and appears much diminished in size.

I trust that the view I have presented of enlargement of the heart, will appear to you to have simplified this important subject. I think, as I advance in the consideration of it, you will find additional reasons for adopting it. It is the practical, the clinical view of the subject. I would not, however, be thought regardless of the different modes in which the heart may increase in size, but surely a diagnosis, which is liable to many exceptions and uncertainties, and which leads to no important practical results, is not a matter of much consequence.*

* Dr. Blackiston of Birmingham, England, has published a table in relation to the different forms of enlargement of the heart, and the comparative frequency with which the right or left side of the heart is affected, founded on 155 post-mortem examinations in the General Hospital of that city. This table, however, is deficient in one important particular. It seems to include in the term dilatation, all those cases in which, the cavities being enlarged, the walls remain of natural thickness, but in which, from the very nature of the case, an increased development of the muscular substance must exist. I judge of this position from the character of the numerous cases reported by Dr. Blackiston, and not from any positive statement he has made. Still, the table is so valuable that I will take some extracts from it. Thus, hypertrophy of both ventricles occurred 44 times. Dilatation of both ventricles 98 times, and, as I suspect, both forms of enlargement were united in a large majority of the cases. Hypertrophy of the left ventricle alone, occurred 20 times, dilatation of the left ventricle 19 times. Hypertrophy of the right ventricle occurred

Enlargement of the heart is met with at all periods of life. It is not uncommon in children, it is frequently met with in adult life, and it is especially common in advanced life. I confess that from the accounts of authors, I was not prepared to meet with the disease so frequently in children and in young adults as I have done. In these cases, if I except those in which there is some congenital malformation of the heart, the almost constant cause is acute articular rheumatism. You will frequently find that the symptoms of heart disease can be traced directly to a rheumatic attack. This will happen in cases in which the seizure of the heart has been violent, so as to have been marked by distinct symptoms at the time, and has produced such decided and permanent disturbance of the functions of the heart as to be rapidly followed by the evidence of enlargement of that organ. But, unfortunately, in how many cases do we want these clear indications of danger to the heart! Too frequently the rheumatic inflammation is very limited in extent, affecting, perhaps, only a single valve, or if more extensive, is masked entirely by the inflammation of the joints. Thus, insidiously, the foundation is surely laid for future disease. This slight and limited inflammation of a single valve may, and often does, become the primary cause of enlargement of the heart, and is the sure forerunner of a fatal issue. If overlooked or neglected, it may produce slowly, but surely, such an impediment to the circulation through the heart, as to lead to incurable enlargement of the organ. Or

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three times only, dilatation 25 times (and as I suspect in many instances, connected in part, at least, with the agony). Thus, to conclude from this table, enlargement of the right ventricle alone may be represented by 28, that of the left ventricle alone by 39, and that of both ventricles together by 142.

In an analysis of 48 cases made by myself,

The right ventricle was affected by		The left ventricle was affected by	
Simple Hypertrophy.....	2 times.	Simple Hypertrophy.....	8 times.
Concentric Hypertrophy....	5 "	Concentric Hypertrophy....	6 "
Hypertrophy and Dilatation	20 "	Hypertrophy and Dilatation	18 "

The above analysis was made many years ago. My subsequent experience has led me to believe that concentric hypertrophy is usually, if not always, dependent upon death from sudden hemorrhage, and is not, strictly speaking, a disease. The tables show, in the clearest manner, the great preponderance of a union of hypertrophy and dilatation, as constituting an enlarged heart, as well as the frequency with which both ventricles are affected together.

attacking the pericardium, it may lead to adhesions interfering with the free action of the heart, and may possibly lead to the same fatal results. I have already in a previous lecture described the changes which rheumatic inflammation, attacking the heart, produces in the pericardium and the endocardium, and their influence in producing enlargement of the heart. I shall again return to the subject when I speak of valvular disease of the heart, in which the remote effects of an obstructed circulation upon the heart are most distinctly observed. The influence of this rheumatic inflammation on the heart is second to none in importance in the whole range of practical medicine, whether it attacks the heart openly, as it sometimes does, or insidiously, as is more frequently the case, so as only to be discovered, and followed in its progress, by the practice of careful auscultation.

The heart is more mechanical in its functions than any other organ in the body, and the causes which lead to its enlargement are for the most part mechanical in their operation. They may be all reduced to this—an impediment to the circulation of the blood through the organ, either from obstruction or from regurgitation. Sometimes the impediment is so near and obvious as to be at once apparent. Thus no one of you can fail to perceive that valvular disease, which obstructs an orifice of the heart, reducing it to perhaps one-half of its ordinary dimensions, or which permits a regurgitation of the blood after it has once been expelled, and thus calling upon the heart for unusual efforts to perform its functions, will lead to enlargement of the organ. But in other cases the impediment is not so obvious. Indeed, you will often find that the orifices of the heart are quite as much enlarged as the body of the organ—the consequence of some remote obstruction. And in this case, if the valves remain of the natural size, the additional evil of regurgitation exists in the heart itself; for if the valves do not enlarge with the orifices to which they belong, their action may be imperfect. You sometimes find the impediment in these cases in the lungs. In the disease known as pulmonary emphysema, the enlarged and rigid air-vesicles press upon and obstruct the pulmonary vessels, so that the heart is called upon for unusual efforts. Hence you will find enlargement of the heart a sure attendant on emphyse-

ma, if it be of long standing and of considerable extent. It has been also supposed that tuberculous disease of the lungs led to the same result, but this is an error. The hearts of those who die of this disease are below the natural average in size. It is true that there is in these cases an obstruction to the pulmonary circulation, quite as great perhaps as in emphysema, but this is more than compensated for by a diminution in the quantity of the circulating fluid. Other remote causes of obstruction no doubt exist, but they frequently escape observation.

It has generally been maintained that emotions of the mind, especially the more violent passions, lead to enlargement of the heart, but I have never seen any evidence of the truth of this assertion. You will read of hearts broken by grief in poems and romances, but you do not meet with them in the matter-of-fact practice of our profession. It was supposed that the horrors of the French Revolution produced a great deal of disease of the heart in Paris. But Corvisart lived at that time, and his labors added so much to the diagnosis and interest of such cases that they attracted much more attention, and were more frequently recognized than before. You are no doubt aware how interest multiplies cases of disease. Thus, let an individual have a disease, and he will at once find out a multitude of those similarly afflicted. So let a new and important mode of treatment for a disease be discovered, and cases seem to multiply at once, simply because they have gained a new interest. But however skeptical I may be as to the influence of the passions in producing enlargement of the heart, I have no doubt whatever that they aggravate the disease when it has once occurred. The passions undoubtedly excite the heart to increased action in some cases and disturb its nervous influence, acting in a way to exhaust it; in other cases, and if organic enlargement has already, from some obstruction to the circulation, commenced, they will certainly hasten its progress. Analogy might lead you to infer that simple increased action of a muscular organ like the heart, from any cause, would lead to enlargement, just as other muscles increase in size by inordinate use. But clinical observation does not confirm this idea. You will see every day, nervous females who have suffered for years from increased action of

the heart—indeed, numerous cases, in men as well as women, in which sympathetic excitement of the heart has long existed, and yet no enlargement has taken place. But if any permanent impediment to the circulation exists, enlargement is usually soon developed. It is the study of these cases, the observation that the heart may act violently and for a long time without enlargement, that has led me to take so mechanical a view of the causes of enlargement of the heart as I have done. And I may add, that these facts are of great importance in the diagnosis of organic from functional disease of the heart. I think that you will find that when the action of the heart has been increased for a long time and no enlargement has ensued, that the disease is functional, not organic.

Advanced age has been regarded as one of the causes of enlargement of the heart. It has been stated as the result of very accurate and extensive observation, that the heart naturally grows larger with the progress of life—that the average size of the organ at sixty years is greater than at forty years; at eighty years than at sixty years. If this is stated as a simple anatomical fact, but half the truth is told. You certainly would not expect this result, when you remember that the muscular system wastes with age. Why, then, should the heart naturally increase in size? I believe that it does not—that it is disease, and not nature, that produces this condition. I shall explain to you in a future lecture that the arteries lose their elasticity with the progress of life, from the almost universal deposit of atheroma. Thus they are less able to assist the heart in carrying on the circulation, an impediment is in fact created, and this tends to induce enlargement of the heart. This enlargement is so slight in very many cases as hardly to be called disease. It is a rather large heart, nothing more. But in other cases, in which the impediment is greater, especially if it occurs near the heart, or at its orifices, then undoubted enlargement ensues, symptoms are present, and death follows.*

* In the Report of the Inspector of the City of New York during three successive years, 721 deaths from "disease of the heart"—a somewhat indefinite expression, intended, I suppose, to include all chronic cases of heart disease, especially enlargement of the organ, with or without valvular disease—are stated: 1 in 64 of

The *rational symptoms* of enlargement of the heart are but two in number, and neither of these are characteristic of the disease. They are palpitation and pain in the precordial region. I have in a few instances met with patients who never, during the whole progress of the disease, had complained of palpitation, and even when questioned carefully and repeatedly, in relation to the fact, they uniformly denied that they were sensible of its existence. I would not be understood to say that, in these cases, there was really no increased action of the heart, but only that the individuals were not sensible of it. It is in this sense that I use the term. In ordinary cases, however, palpitation marks the onset of the disease—at first being slight, and only noticed after some unusual excitement, but gradually becoming more frequent and distressing. In this respect, it differs from the purely nervous palpitations you will frequently be called to witness. Such cases often occur suddenly, and with great violence, and leave the patient quite as rapidly. The frequent occurrence of palpitation, as a mere nervous symptom, and its permanence also, in many cases when the exciting cause continues, will prevent you from regarding it as at all characteristic

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the general mortality. Of these 399 were males, and 322 were females. In 710 cases, 165 were under 20 years of age; 213 between 20 and 40 years; 332 over 40 years of age.

From June to December ..... 350 died.
From December to June .....372 "

The relative mortality in London from "disease of the heart" during eight years (1840 to 1848), to the general mortality, was 1 in 37·3 deaths.

From October to April .....5910 died.
From April to October .....4737 "

The increase of fatal cases during the winter and the spring months, both in London and in New York, is probably owing to the more frequent complication of severe and sudden inflammatory and congestive pulmonary attacks.

Statement showing out of 100,000 persons born in London, the numbers dying of heart disease, the sex, and the relative ages:

Males: of 51,023 born, 135 die before the 15th year; 268 from 15 to 40 years; 933 after 40 years of age.

Females: of 48,977 born, 107 die before the 15th year; 194 from 15 to 40 years; 908 after 40 years of age.

Of these 100,000 persons, male and female, there are alive at 20 years, 61·6 in 100; at 40 years, 49·8 in 100.

of enlargement of the heart. It may, however, be stated as a general fact, that nervous palpitation is much more intermittent and irregular in its existence than that produced by organic disease. In the latter case, it may, indeed, be absent for a time, but the same amount of excitement will infallibly induce it; while in merely functional disturbance, a slight excitement will sometimes develop it with great violence, while at other times, and in the same patient, a much greater degree of excitement will not develop it at all.

Pain in the precordial region is a frequent, but by no means constant, symptom of enlargement of the heart. I have occasionally met with patients who had not complained of palpitation, while absence of pain is still more common. Often the sensation complained of, is a sense of weariness or ill-defined uneasiness, resembling that experienced during the existence of grief, or after a paroxysm of anger. Sometimes it is a sensation of fulness, or of sinking, referred more frequently to the epigastrium, or to the lower portion of the sternum. Occasionally, it is a sense of heat, as if the heart had been dipped in hot water. Finally, sharp pains sometimes strike through the heart, and extend even across the chest and down the arms, especially the left arm. When you recollect that attacks of partial pericarditis are not unfrequent in enlargement of the heart, you will find a ready explanation for the pain in some cases. In most cases, however, it is evidently neuralgic in its character, while in other cases it is, no doubt, the result of the oppression and labor that the heart is compelled to undergo.

Pain in the precordial region sometimes assumes an intensity, and is sometimes accompanied by other symptoms so peculiar and striking, that it is usually described as a distinct disease, under the name of *Angina Pectoris*. It is in most cases associated with organic disease of the heart, and I have found it existing in its worst form when nothing but simple enlargement of the heart was noticed after death. This condition of the heart being most frequently found in connection with the symptoms of angina, I shall describe its symptoms in connection with that form of disease. Still, I am ready to admit that other conditions of the heart are frequently present, especially such as

tend to weaken the muscular power of the organ. Many cases of angina, for instance, are connected with the deposit of fat upon the heart, attended by an atrophy of the muscular fibres, or upon the peculiar degeneration of the muscular fibre itself into fat, conditions which may or may not be connected with an enlargement of the heart. This fatty degeneration of the heart has been found to be not unfrequently associated with ossification of the coronary arteries, and is supposed to be dependent, in part at least, upon the imperfect nutrition of the organ from this cause. This fact curiously connects itself with the original observations of Parry on Angina Pectoris, the first author, I believe, who attempted to connect its symptoms with a particular form of organic disease, viz., ossification of the coronary arteries. In other cases, the walls of the heart may be simply more thin or soft than is natural.

But as cases of angina sometimes recover, the affection cannot necessarily be regarded as connected with any permanent form of organic disease of the heart. Still there is probably always, even in functional cases, a feeble heart. I shall call your attention, in another lecture, to the affection as connected with functional disturbance of the heart.

The symptoms of angina pectoris are not constant, but paroxysmal, and are almost invariably produced by some recognized excitement, physical or mental—usually the former. While the patient is quiet and tranquil, he does not suffer. But if he walks, or rides on horseback facing a strong wind; if he goes hastily up stairs; if a degree of mental anxiety is combined with even a less degree of physical effort, as, for instance, if the patient is searching for an individual, or a place he is anxious to find; if he gets into his carriage rather hastily, with the fear that he has kept a friend waiting too long—then a paroxysm may occur. In the more advanced cases, even slighter causes will induce it; as stooping to tie a shoestring, turning in bed, a flatulent state of the stomach, a little mental excitement. Finally, the disturbed sleep, which these unhappy patients get only at intervals, is sometimes interrupted by a paroxysm.

A paroxysm of angina, according to my observation, is most frequently marked by a sensation of distressing, death-like sink-

ing, which is referred to the lower, or sometimes to the middle portion of the sternum. Ask the patient to describe the feeling —ask him if he feels pain. He will usually answer no; but that he experiences a dreadful sinking sensation, as, if it did not at once stop, he must die. Sometimes, however, there is excruciating pain in the heart, as if the organ was grappled with iron claws, and shooting across the sternum down the left arm, along the track of the nerves. In severe cases, the pain may extend down both arms, or pass through to the back, and extend down the lower extremities. In these paroxysms there is often a sensation of numbness with the pain—sometimes a sensation of weight; and I have noticed a blueness about the fingers. Sometimes, however, the pain in the extremities is excruciating, tearing, like that in the heart. The face is usually pale, the respiration labored and somewhat accelerated, and the pulse becomes more rapid, weak, fluttering, and sometimes extinct. As the paroxysm subsides, as it usually does after a few moments of rest, and sometimes with the copious extrication of gas from the stomach, the pulse becomes more full and slow, the respiration more tranquil, and the patient, rubbing his hand across the chest, complains of nothing but a tired sensation. Sometimes reaction goes beyond the mark; and a temporary tumultuous action of the heart may ensue.

Now, what is the cause of the paroxysm? That it is neuralgic in its character there can be but little doubt, at least so far as pain is concerned. But pain, in its proper sense, is not present in perhaps a majority of instances. It is sinking, "dreadful sinking," that is complained of; and this is attended, not with syncope, although there is diminished action of the heart, and even pulselessness. The patient sits up, or, if standing, leans against some support, but does not fall senseless, unless the paroxysm terminates, as it sometimes does, in sudden death. The explanation usually offered of these phenomena is, *congestion of the heart.* Thus, the heart, enfeebled by disease, and disturbed in its action, has its cavities suddenly filled with blood which it cannot expel. The mechanism of this congestion is not alike in all cases. Under the influence of some momentary excitement of the circulation in some cases, the heart receives more blood than

it can easily get rid of—under the influence of a temporary and increased obstruction to the circulation in other cases, the same result is produced. Thus you can readily see, on the one hand, how a heart that has just vigor enough to maintain a tolerable circulation through its cavities, may, by being pushed a little too far by excitement, receive more blood than it can dispose of, and that this distension, by paralyzing the muscular power of the heart, may add to the difficulty; so, on the other hand, the slight obstruction of the circulation, from stooping forward and thus compressing the aorta, or from the accumulation of flatus, may overpower the weak heart in its efforts to expel the blood, and lead to a similar congestion of its cavities.

But if this be admitted as the general explanation of the paroxysm, it does not explain all the phenomena. It explains the feeble pulse, the sense of sinking and distress, perhaps, but it does not explain the existence of pain, which is often of the most severe character, and evidently neuralgic. I confess, I know of no explanation that is at all satisfactory: many explanations have been offered, but none of them are conclusive. It is easy, however, to explain the cause of sudden death, by no means an unfrequent occurrence in these cases. The congestion carried too far, paralyzes the muscular power of the heart by distension, so that it cannot recover its action.

In cases, also, in which there is no positive organic disease of the heart, but only a weak heart, you can readily recognize an important element to favor congestion. But in this case also, there is something beyond, that is not so clear. A feeble heart is common enough from a variety of causes, and yet, you will seldom find with it the phenomena of angina pectoris. There must, then, be something else to induce the symptoms of angina in cases in which they occur; perhaps remote disease, which escapes observation. But I do not wish to occupy your time with uncertainties. I will therefore pass from this subject, remarking, however, that there are many points in heart diseases, as in all other diseases, that will perplex you exceedingly, when you seek their full explanation.

A gentleman, aged 43 years, and who had generally enjoyed excellent health, was suddenly attacked at night, after rising to

open a window, with a severe paroxysm of suffocation. These paroxysms continued to recur, at intervals, and, in the course of a few weeks, assumed the characters of angina pectoris. I saw him soon after this time, and about four months after the first attack of dyspnœa. His countenance was pale, his nervous system was excited, and his pulse accelerated; he suffered from habitual dyspnœa, and had lost some flesh.

His paroxysms of angina were marked by a "sinking sensation," extremely distressing, referred to the middle of the sternum, and were accompanied by a sense of suffocation and by labored respiration. Twice he has experienced, during the paroxysm, a sharp pain darting through the heart, like a knife; once it occurred after stooping. There has been no palpitation.

The heart was evidently enlarged, the apex striking too far to the left, but the precordial dulness was not much increased. The action of the heart was moderate. There were no morbid sounds noticed.

This case progressed steadily to its termination, about six months after the first attack. After about a month, considerable effusion occurred into the right pleural sac, and this was followed by anasarca of the legs, and by slight ascites. The most distressing symptom was dyspnœa, and the attacks of sinking, as he described them, recurring almost daily, were sometimes attended by trifling pain about the heart, but not often. The action of the heart was feeble, and often irregular. The patient, after repeated and careful inquiries, always stated that he had never experienced the slightest palpitation.

After death, considerable effusion was found in the right pleural cavity. The heart was generally enlarged, and of good consistence. There was no valvular disease. No disease was noticed in other organs.

The cause of obstruction to the circulation was not apparent. It existed somewhere remote from the heart. Here, again, dyspnœa was the first, and continued to be the most prominent symptom, and yet the right side of the heart was not more affected than the left side. The lungs did not present any evidence of primary obstruction to the circulation, yet, as dropsical effusion first occurred in the pleural cavity, and dyspnœa was

the first symptom, the true seat of the obstruction was, probably, in these organs.

I may remark, also, that the action of the heart was feeble during the whole time I attended him—three months after the attack. Yet the patient was a man of stout frame, free from previous disease, and the muscular structure of the heart was found of good consistence after death. I can only refer the feebleness of the heart's action, in this case, to impaired nervous energy; a condition which, I believe, operates powerfully, in many cases of enlargement of the heart, to diminish its action, and to predispose to dropsical effusion, and, as in the present case, to angina pectoris.

A gentleman, aged forty-nine years, of a vigorous constitution, had been ill for about eighteen months with symptoms of disturbed circulation, and for several months the symptoms of angina pectoris had recurred in frequent paroxysms.

The patient, a very intelligent man, described his paroxysm as coming on, especially after taking food, or using exercise. The first sensation was a distressing sinking at the epigastrium, followed by pain in the same region, increasing in severity, and compared to a tired, aching feeling. It extended up to the neck, and then down the left arm; it was especially felt in the fingers. There was no lividity of the countenance, but rather paleness. The respiration was not sensibly affected, neither was the pulse, according to the statement of the patient. Complete relief always followed the extrication of gas from the stomach.

On examining the precordial region, the heart was found evidently much enlarged. There was increased dulness and dilatation over the precordial region, and the apex struck between the sixth and seventh ribs, under the left nipple. There was a double, blowing sound over the aortic valves most distinctly masked with the second sound of the heart, indicating both aortic obstruction and regurgitation. The impulse was moderate, the rhythm natural, the pulse was accelerated, quick, and jerking.

This patient died suddenly, after an interval of about two months. No post-mortem examination was made.

A gentleman, aged about sixty years, and habitually in the

enjoyment of good health, although of rather delicate constitution, and somewhat dyspeptic, had noticed for two years dyspnœa and distress in the precordial region after sudden exertion. He had, also, for some time, suffered from palpitation, but had continued his business, that of an active merchant. He has, also, had one or two attacks resembling angina pectoris. On examining the heart, I could find no decided evidence of enlargement, but there was a distinct, rough, blowing, and rather musical sound heard over the aortic valves, and extending up along the aorta. It was less distinct over the mitral valve, but grew more marked over the body of the left ventricle, and was heard very distinctly at the apex. It masked the first sound of the heart. The action of the heart was regular. Intending to call on me one evening, soon afterwards, he was somewhat perplexed and hurried in finding the right door, so that he had a paroxysm of angina as soon as he entered the room. He complained of the same distressing symptom—sinking; the respiration was evidently labored, the countenance was pale and distressed, and the pulse was very feeble and rapid. In a few moments the paroxysm subsided, and he was disposed to rub the front of the chest with his hand, and complained of a tired feeling. The pulse became more full and slow, and considerable reaction ensued. The heart, examined immediately after the paroxysm, presented the same physical signs as at the former visit.

I mention this case because I saw the patient during a paroxysm of angina, and have described the symptoms as I noted them at the time. He lived, perhaps, a year after this time, and died rather suddenly.

LECTURE XXIX.

ENLARGEMENT OF THE HEART.

Physical signs; displacement of the apex—extended impulse; increased precordial dulness on percussion—dilatation.—Constitutional symptoms; dyspnœa, hemorrhage, dropsical effusions, bronchitis; pulse.

In my previous lecture I remarked that the rational signs of enlargement of the heart are not characteristic. This is not true of the *physical signs*. Taken together, they are highly characteristic, and will safely lead you to the accurate diagnosis of even a moderate enlargement of the organ. The great importance of this fact will be more apparent to you, when you recollect that enlargement of the heart is the starting point of many cases of organic disease of this organ, and the direct and sure consequence in the remaining cases. For if the attack begins in the valves, or in the pericardium, enlargement of the heart is the result, and the establishment of its existence becomes the surest basis of the diagnosis of organic disease from functional, mere functional derangement of the organ. To be able to say, then, that a patient's heart is enlarged, or not, is giving a decision which often condemns him to death, or at best makes an invalid of him for life; or, on the other hand, affords him the prospect of a long life, and even of a speedy recovery.

The physical signs, like the rational symptoms of enlargement of the heart, are but few in number. The most characteristic is the point where the apex of the heart strikes against the ribs. You will recollect that while speaking of the physiological action of the heart, I remarked that the apex of the heart strikes the walls of the chest between the fifth and the sixth ribs, below, and to the inside of the nipple. When you consider the position of the heart, lying obliquely across the chest and attached by its base only, you will easily understand how any enlargement of the ventricles (unless the disease should be confined to the right ventricle, and then not always) would cer-

tainly displace the position of the apex by carrying it further to the left, as well as downward. The attachment of the pericardium to the diaphragm does not appear to interfere with this result. How many times have I ascertained the existence of enlargement of the heart by simply finding that the point where the apex struck was between the sixth and the seventh ribs, perhaps quite to the outside of the nipple! In a few cases, especially of excessive enlargement, I have found the apex striking above the line of its natural level—but far to the outside, to the left of its natural position. In these cases you will find the heart, after death, lying transversely across the chest, its apex being carried upward. Generally, it is easy to find the point where the apex strikes, both by the touch and by the eye. You might suppose that it would be easier to ascertain this point in the enlarged than in the healthy heart, but it is not always so. It would be a great mistake to suppose that in every case of enlargement there is strong action of the heart. In many cases, perhaps in most cases of advanced enlargement, the action of the heart is feeble. The heart becomes worn out, if I may so speak, and, like a tired muscle, acts feebly; so that if you feel for the apex, you may not find its striking point. There is another reason why this may happen. The heart as it enlarges fills up the mediastinal spaces, so that there is no longer any room for the apex to spring forward against the ribs—the whole surface of the heart lies against the walls of the chest. Thus, in cases of enormous enlargement, even when this is chiefly hypertrophy of the muscular substance, there is rather an obscure heaving than a distinct impulse. Again, the unusual presence of fat or of muscle in the precordial region, especially the presence of the mamma in women who have borne children, as well as the feeling of delicacy which may interfere with your exposing the chest in all cases, are obstacles to the ascertaining the precise position of the apex when the action of the heart is feeble. Finally, effusions of liquid into the pericardium, by pushing back the heart, may prevent you from finding the apex. In acute effusion the delay would probably be but temporary, the absorption of the fluid would soon permit the heart to return to its proper place; and chronic effusion,

or dropsy of the pericardium, is so exceedingly rare in my observation, that it may almost be left out of the question. Chronic adhesions, however, the result of former pericarditis, by which the opposite surfaces of the pericardium adhere together, do interfere with the striking of the apex against the ribs, and if unusually thick, may entirely prevent its being felt. In those cases in which I cannot readily feel the point where the apex strikes, I use the solid stethoscope, placing it upon the natural position of the apex, and then gradually passing to the left until I find the precise spot where the shock of the heart ceases.

The circumstances I have just mentioned are causes of difficulty, but there are other circumstances which may lead you into error. It will not be safe when you find the apex of the heart displaced in the manner I have mentioned, to say at once that the heart is enlarged. The heart is liable to displacement from other causes besides its own growth. The most common cause is an effusion of liquid into the cavity of the right pleura, which, if considerable, may push the heart to the left side. An enlargement of the left lobe of the liver may do the same thing. Even an accumulation of gas in the stomach, or of fluid in the abdominal cavity, may displace the heart, pushing it upward at least, if not to the left. Indeed, the position of the patient, if he be lying down and inclined to the left side, will produce a slight displacement to the striking point of the apex, which might be mistaken for an evidence of enlarged heart. A patient, with suspected disease of the heart, should, as I have already stated, be examined either in a sitting or in a standing position. There are but few cases in which this is not possible.

When the heart is much enlarged, you may find an absence of respiration over the precordial region, as well as increased dulness on percussion. But these signs are of far less importance than the position of the apex, as you will readily perceive. In the first place, if the chest be largely developed, and the lungs large and active, they may still cover the enlarged heart so that no unusual dulness on percussion, or absence of respiratory murmur, may be noticed in the precordial region. Again: in other cases, very few indeed, these changes may be owing to

a cause residing in the heart itself, but entirely independent of enlargement—effusion of fluid in the pericardial sac; or there may be dulness from effusion into the cavity of the pleura, too extensive, however, to be referred to the heart. It is common, nevertheless, to find an increased dulness on percussion, both in extent and in degree, over the precordial region, in cases of enlargement of the heart. In the healthy state of the chest, you will recollect that the dulness is limited to about two inches square, and that this is a comparative, not an absolute, dulness; so that considerable practice and tact in percussing the precordial region, is requisite to fix a just value to this sign. The method of ascertaining the size of the heart by the mode of percussion introduced by Drs. Cammann and Clark, is much more accurate than that already mentioned.

A dilatation of the walls of the chest over the precordial region is, sometimes, the direct effect of an enlargement of the heart. Effusion into the pericardium will produce the same effect. But a greater source of error is the frequent malformation of the walls of the chest. If you carefully examine the chests of a large number of individuals, you will be surprised to find how few are perfectly symmetrical, and how frequently there will be noticed a slight bulging or depression, residing entirely in the parietes. These slight deformities cannot strictly be regarded as disease; they lead to nothing. Now one of the most frequent seats of this bulging is the precordial region, and the fact should be remembered, that you may not mistake its true character. If it be united to unusual dulness on percussion, absence of respiration, displacement of the apex, and other signs of enlargement of the heart, you may fairly attribute it to this cause. Another circumstance, however, must not be forgotten. When dilatation of the parietes is owing to internal pressure, the intercostal spaces are pushed out as well as the ribs, which is not the case when the cause of the deformity is in the parietes.

When you apply the stethoscope to the region of an enlarged heart, you will feel the impulse over a surface more extended than natural, and proportioned to the degree of enlargement. But let me caution you. Do not mistake the general jar of the

walls of the chest for the true impulse. A heart of natural size, acting with unusual force, especially in a narrow chest with thin parietes, will jar the whole chest, and particularly the precordial region; so that even a delicate finger shall hardly distinguish the true impulse from the general jar. By the region of the true impulse, I mean that portion of the chest that is directly struck by the heart during its systole, and this can be most satisfactorily ascertained by the use of the solid stethoscope. When you apply this instrument exactly over the heart, the ear receives a shock, which appears to come from directly under the instrument. When the instrument is applied beyond the region of the heart, you perceive, indeed, a diffused jar, which reaches you from a distance, but does not appear to come to the ear from directly under the stethoscope. A practical point like this is much better explained at the bed-side than in the lecture-room. Yet its importance has, I think, justified me in dwelling upon it. Important errors in diagnosis are frequently made by mistaking the general jar of a merely active heart from the extended impulse of an enlarged one.

Again, you will frequently find, in this disease, that the impulse of the heart is increased in force, but this sign is of far less importance than the extended impulse I have just alluded to, and for several reasons. In the first place, a heart may have its impulse much increased in degree, and yet be entirely free from organic disease. You see it illustrated every day in the simple effect of active exercise. In many nervous subjects, especially when the heart suffers sympathetically from some remote irritation, its action may become even highly tumultuous. In the early stage of organic enlargement of the heart, the impulse is almost always increased in degree, especially when there is much hypertrophy of the walls of the left ventricle. But even in these cases, the time will probably come when the impulse will gradually diminish, and very likely, during the more advanced period of the disease, it will be even less than is natural. Placing the hand over the heart, in these cases, you may feel, almost absolutely, no movement, and yet the heart may be enormous in size, and the enlargement be principally of the form of hypertrophy. The cause of this is threefold: First,

the vital powers generally have now become much impaired, and the power of the heart fails with the rest. Secondly, the heart seems to wear itself out by its long-continued activity—like other tired muscles, it loses its vigor and acts feebly, often irregularly. Finally, as it enlarges, it becomes pent up against the walls of the chest, and can no longer spring forward with force. These remarks apply even to those cases in which the enlargement is chiefly owing to muscular hypertrophy: how much earlier and more decidedly should this apply to those cases in which the enlargement exists, principally, in the form of dilatation of the cavities. I am inclined to think, also, that the adhesions of the pericardium to the heart, which not unfrequently exist in connection with enlargement of that organ, tend to diminish the force of the impulse. Let me now repeat the conclusion I would arrive at, and impress it upon your minds. Impulse increased *in extent* is an important sign of enlargement of the heart; impulse increased *in degree* means, necessarily, nothing.

Irregularity in the rhythm of the heart has been regarded as an evidence of disease, but this is not necessarily so. I have known persons whose pulse has been irregular for years, and who never suffered from any symptoms of disease of the heart. Temporary excitement will produce it in nervous persons, and so will remote local irritation. As a symptom of enlarged heart, it is not of much importance. It very frequently exists, especially in the advanced stage, when the nervous energy of the organ is impaired. It may be constant, but more commonly it occurs at intervals. Sometimes it presents itself by intermissions in the beats of the heart, at other times the beats are irregular in frequency and in force. Indeed, an observer disposed to be over-nice in the matter, might find many curious varieties in the irregular action of the heart, but they would lead, in the present state of our knowledge, to no practical results. I have noticed this irregular action to be produced by precisely opposite exciting causes. One patient will have it induced by exercise, another by quiet.

When you listen to the *sounds of the heart* during a state of enlargement of that organ, you will often discover nothing re-

markable. When the enlargement is general, and consists, about equally, of hypertrophy and of dilatation, and this is the most common case, the sounds will appear natural, or at most, a little exaggerated. In cases in which hypertrophy predominates, especially if excessive, the sounds become more dull than natural, owing to the *clear* element of these sounds, if I may so speak—that residing in the tension of the valves, having to reach the ear from a greater depth, and after passing through a greater thickness of structure; while in cases of dilatation, the thinness of the walls brings out, as it were, this valvular element, and makes the sounds more clear and sharp than is natural. This is particularly true in relation to the first sound of the heart, which, as I have remarked in a previous lecture, is compound in its character, being produced by the tension of the mitral and tricuspid valves, the impulse of the heart against the ribs, and, perhaps, by muscular tension united. Now in dilatation, the influence of the two latter elements is much impaired, while the former is exaggerated, so that in fact you may sometimes find a difficulty in distinguishing the first sound of the heart by its tone, from the second sound, so valve-like has it become. Thus it is that disease comes to the aid of physiological experiment in illustrating the mechanism of the sounds of the heart.

The influence of enlargement of the heart upon the *constitution*, and upon *remote organs*, is very striking and important. Regarded singly, none of the symptoms thus produced can be considered as characteristic; but if studied in their order of succession, and in connection with each other, they will furnish you with important aid in the diagnosis. I may state, as a general fact, that in the early stage there is a tendency to active or arterial congestion, leading, not unfrequently, to hemorrhage of different organs; while in the later stage there is a tendency to passive or venous congestion, leading, almost invariably, and sooner or later, to dropsical effusion. This general fact is the key to many of the symptoms connected with an enlarged heart, and should be deeply impressed upon your memories. It follows, from what I have already stated, that in the early stage of the disease you are considering, the action, the power

of the heart is increased, while in the advanced stage it is diminished.

In this disease, the general powers of the constitution do not fail rapidly, as a general rule. Often, when it is well developed, the general muscular strength remains unimpaired, and the flesh does not waste. Sometimes, indeed, it imparts a freshness and color to the countenance, which might easily be mistaken for florid health. But in time the general strength diminishes, emaciation ensues, although often disguised by dropsical effusion, and the countenance assumes a pale, livid, heavy, anxious expression, which is highly indicative of the disease. Febrile excitement is not an attendant on any stage of enlargement of the heart, unless induced by some inflammatory complication, although the pulse is usually found rather accelerated.

The lungs are so intimately associated with the heart, and the disturbance of their functions is so constant and so prominent when the heart is enlarged, that, for practical purposes, I might have considered dyspnœa among the rational symptoms of enlargement of the organ. But this arrangement would have been wanting in scientific precision, since the lungs, however intimate their connection with the heart, are yet distinct organs, with distinct functions. Still, let the above remark impress upon you the very important and prominent part that dyspnœa plays in enlargement of the heart, and my object will be gained. Indeed, so prominent is this part, in many instances, that numerous cases go through their whole course under the eye of the physician, without their true nature being suspected. They are called, in vague language, cases of asthma, and so completely is the attention taken up with the dyspnœa, that the heart is never thought of. Many cases of enlargement of the heart are ushered into notice by a violent paroxysm of dyspnœa, which may be repeated, or subside into a steady oppression of the respiration. But, as a general rule, dyspnœa comes on gradually, like palpitation; and although a severe paroxysm, induced by some unusual exciting cause, may first attract the serious attention of the patient, yet careful inquiry will usually ascertain the fact, that the symptom has existed in a less degree before, and has even been slowly developing itself for a long time. Even

among the more intelligent, a symptom may not attract attention, or be forgotten, if it does not interfere with the ordinary business and comfort of life, and especially if it be only an exaggeration of what is natural. Thus, a little more dyspnœa in going up stairs, or in walking, is unnoticed, while a violent effort, as running up a hill, or in the face of a strong wind, arrests the patient so suddenly, and occasions so much distress, that he at once takes the alarm.

There is nothing characteristic in the dyspnœa of enlargement of the heart, although its constancy under the same circumstances is its most important feature—that is, at first it constantly occurs after the same degree and kind of excitement, as that, for instance, of going up a flight of stairs, while when the patient is quiet nothing of the kind is felt. In these cases it is simply the increased action of the heart, forcing the blood more rapidly through the lungs, and thus rendering a more rapid oxygenation necessary, that induces a more full and rapid action of the respiratory function—or dyspnœa. The same result is noticed every day in health, when exercise, by increasing the action of the heart, increases the respiration also. But as the disease advances, new and very different causes of dyspnœa ensue. It is no longer the rapid passage of the blood through the heart that causes dyspnœa, but the reverse—an obstruction to its passage from enfeebled action. New causes of dyspnœa are also added in the supervention of bronchitis, pulmonary œdema, effusion of serum in the cavities of the pleura; so that, in advanced cases of simple enlargement of the heart, the dyspnœa usually becomes not only a constant, but a most distressing symptom. The least exertion brings on a paroxysm—the patient being compelled to sit in his chair for days and for nights together, often gasping for breath, until, after a long agony, death happily puts an end to his sufferings.

Irritation of the bronchial tubes almost constantly occurs in the course of the disease, not usually at an early period, but rather after it is fully developed. It has appeared to me, that the disordered circulation in the lungs impressed a peculiar irritability upon the bronchi, indicated by a cough, either dry or attended with but little expectoration, and especially by the sib-

ilant and sonorous rhonchi diffused over the chest. This condition tends to aggravate the dyspnœa, and adds materially to the general distress. When it has once occurred, it is usually permanent. Under the influence of ordinary exciting causes, as exposure to cold, it may pass into a severe acute bronchitis, attended by much mucous secretion, with a mucous rattle over the posterior portions of the chest, and assume the features of what may be called a suffocative catarrh—the dyspnœa becomes intense, orthopnœa even may ensue, the countenance assumes an unusually livid tint, and asphyxia may supervene. Death, indeed, is not an unfrequent result of such an accident.

During the early and active period of enlargement of the heart the countenance is often flushed, with increased heat in the head, attended by pain and throbbing in the temples. Such cases often end in apoplexy, the result of active or arterial congestion. It is very common for the true nature of such cases to be entirely overlooked. There is so little disturbance of the functions of the heart, and so much more prominence and inconvenience in the affection of the head, that this is easily accounted for. Attention, once drawn to the heart, will detect the primary disease; but it frequently happens that an examination after death first reveals the enlargement of the heart, which is often an hypertrophy of the left ventricle. A very considerable proportion of the cases of apoplexy in middle life occur in this way.*

A surgeon of the navy, aged about forty-five years, and of rather delicate constitution, had for a long time supposed that he was affected with some disease of the liver. He had also, for a long time, complained of pain in the head, and had experienced palpitation and dyspnœa in ascending elevations. Some months before his death, while operating upon a patient, he felt the sensation of numbness in his arm, which, however, soon subsided. Two days before his death, and after complaining of his head more than usual, he experienced, while in bed, a sensation of

* In seventeen cases of cerebral hemorrhage reported by Andral in his Medical Clinic, nine exhibited hypertrophy, especially of the left ventricle of the heart, and in all these cases the aortic orifice was free.

numbness in his left arm, and in attempting to rise he found that the whole left side was paralyzed. The next day the muscles of the face were also noticed to be paralyzed, but the pupils were natural. He died apoplectic, twelve hours after the attack.

By post-mortem examination, a large clot was found in the centre of the right hemisphere of the brain, extending into the right ventricle. A thin layer of cerebral substance around the clot was softer than natural, and of a slightly yellowish tint. The membranes and the other portions of the brain were natural. The arteries were not ossified. The left ventricle of the heart was hypertrophied. Nothing else abnormal was noticed in the heart, except a few atheromatous spots upon the mitral valve. The lungs, liver, spleen, stomach, and pancreas were healthy.

This tendency to apoplexy sometimes occurs at a much more advanced period of life, when the arteries of the brain may be supposed to be diseased, especially when extensive atheromatous deposits exist in the aorta.

A man, seventy years of age, was brought into the New York Hospital in a state of insensibility. He had been accidentally found in this condition. He breathed with great difficulty and with stertor, and the right side of his body was paralyzed. The pupils were immovable.

Post-mortem examination disclosed a large clot of blood in the left hemisphere of the cerebrum. The substance of the brain was broken down into a cavity capable of containing a large orange. The heart was much enlarged. The aortic valves were bone-like, and bone-like patches extended down the aorta as far as the diaphragm. The lungs and the liver were healthy.

As the disease of the heart advances, and obstruction to the circulation becomes the predominating tendency, apoplectic attacks are less frequent, but there is no immunity from them. The head still suffers, but serous effusion may be more naturally expected. There is sometimes, in this disease, a peculiar irritability of temper developed, which, like the irritation of the bronchi I have mentioned, appears to me to depend upon a disordered circulation. It stamps itself upon the countenance by a look of anxiety, it shows itself in fretfulness, and sometimes in violent paroxysms of fury resembling insanity. The influence of the

heart on the emotions is by no means a mere fancy, neither is the reaction of the emotions upon the heart an idle tale. No doubt many cases of disease of the heart are aggravated by this unusual excitability of the temper, and even many cases of sudden death are caused by an uncontrollable burst of passion. The influence of these views on the proper management of such cases, in prolonging life, must at once be apparent to you.

Active congestion of other organs may occur and hemorrhage ensue. Thus, the mucous membrane of the nose may become congested, and epistaxis follow, with a most happy relief to the brain. Or pulmonary apoplexy, often attended by hæmoptysis, may ensue from hypertrophy of the right ventricle. In pulmonary apoplexy, however, there is commonly another cause more prominent, and, I think, earlier in its development, and this is a narrowing or imperfect closure of the left auriculo-ventricular orifice. Here the difficulty commences; the blood is checked in its return from the lungs, congestion of the left, then of the right portion of the pulmonary circulation, ensues, the right ventricle becomes enlarged with the effort to overcome the resistance. Thus, between obstruction on the one side, and increased action on the other, pulmonary congestion and apoplexy ensue.

But this obstruction to the return of the blood from the lung from disease of the mitral orifice, is not necessary to produce pulmonary apoplexy. It may occur in connection with simple general enlargement of the heart.

A seaman entered this Hospital who had been subject for many years to attacks of miasmatic fever. About a year before admission, he had an attack of pleurisy. For several years he had been subject to palpitation and to attacks of bronchitis, and for a year past he had suffered from a constant cough, with free expectoration frequently tinged with blood. Of late, he had been subject to night-sweats and to other symptoms of hectic. Two days after admission he expectorated two ounces of blood. On examining the chest, the respiratory murmur was generally feeble, and a marked sonorous rhonchus existed all over the lungs. The region of the lungs sounded well on percussion; while over the precordial region there was marked dulness on percussion, but no dilatation. The apex struck outside the left

nipple, and between the sixth and the seventh ribs. The impulse of the heart was strong and labored. The systolic sound was dull, the diastolic sound was clear.

In the progress of the case, the palpitation and dyspnœa were very severe. The action of the heart was tumultuous, and a blowing systolic sound was detected over the aortic valves. The action of the heart became irregular, there was a tendency to faintness. Delirium ensued, and death took place after great suffering from dyspnœa.

Post-mortem examination disclosed large patches of pulmonary apoplexy in the lower lobes of both lungs. The heart was very much enlarged. The spleen was also enlarged, the liver was healthy. The brain was found in a natural condition.

I do not think that active congestion plays an important part in the abdominal organs in this disease. Hemorrhage, at all events, is not common. May not this be owing to the subordinate part which the arterial system plays in comparison with the venous system in this portion of the body? The effects of venous, or passive congestion, are usually very evident in the advanced stage of the disease, in the production of enlarged liver and spleen, fulness of the venous branches, as noticed after death; and especially in the appearance of ascites.

Serous effusion, as I have already stated, is a symptom of the advanced stage of enlargement of the heart, when the heart, as well as the vital powers generally, are more or less exhausted. It is the immediate effect of enfeebled venous circulation, and is first noticed in those parts in which this circulation is naturally most difficult, that is, in the feet and about the ankles; the obstructed veins trying to relieve themselves by pouring out the serous portions of their blood. This symptom is so constant, that very few who suffer from this disease escape it, unless suddenly cut off from life. It may occur earlier, in many cases, than you might expect, from the existence of accidental causes, as valvular disease, acute bronchitis, effusion into the chest from acute pleuritis, or from any sudden cause inducing an increased obstruction to the circulation. Dropsy in the lower extremities cannot be regarded as a characteristic symptom of diseased

heart. It may be the result of simple debility, and is often noticed during the convalescence of exhausting acute diseases, as typhoid fever. It may occur in cases of simple anemia, or in chlorosis, and here, as there is often much functional disturbance of the heart, its true character might be more readily mistaken. It occurs also towards the close of many chronic debilitating diseases, especially in tuberculous phthisis. Still, when you find dropsy occurring first in the feet, and gradually increasing by extending up the lower extremities, followed by ascites and perhaps by hydrothorax, and if this has been long preceded by heart symptoms, you can have a strong presumption as to the true cause, but not an absolute certainty until you examine the heart and ascertain its true condition. I may mention a fact of much practical importance in the diagnosis of dropsy from organic causes. Observation will enable you to recognize three distinct forms of organic dropsy; that from heart disease, in which I believe the effusion commences in the feet; that from some organic cause obstructing the portal circulation, of which cirrhosis of the liver is the most frequent, in which the effusion commences in the abdomen; and, finally, that from Bright's disease, in which the effusion commences in the face and in the sub-cutaneous cellular tissue generally. When called to attend a case, you may find the effusion in all these parts, and it may be difficult, if not impossible, to unravel the order of occurrence; but when it can be done, I think you will find the general rule as I have stated it. You may meet with occasional exceptions.

The progress of the dropsical effusion in enlargement of the heart is upward, from the cellular tissue about the ankles to that of the thighs and scrotum, then to the cavity of the peritoneum, and finally to the cavities of the chest. This order of succession will aid you, often and materially, in the prognosis; for when the chest is affected, the chances even of relief are much diminished. In my experience, serous effusion is not often removed from the chest in heart disease. It is a common opinion also that effusion into the pericardial sac is of frequent occurrence in these cases, but I have found it exceedingly rare. I do not remember to have met with more than two cases in ten years. It

is true, that during the prolonged and mortal agony that often attends this disease, a considerable effusion of serum is poured out into the pericardial sac, and found there after death. So the effusion into the pleural sacs is increased, and may even originate in many cases at the last moments of life. But an effusion into the pericardium, occurring during the advanced period of the disease, and continuing long enough to become the subject of careful examination, is very rare. Dropsical effusion sometimes takes place in the lungs, either into the air-cells, or into the common cellular tissue. In the former case, you will notice increased dyspnœa and a fine crepitation masking the respiratory murmur and heard more or less extensively over the chest. In cases in which the common cellular tissue is the seat of the effusion, you will observe the same increase of dyspnœa, but no crepitation; the respiratory murmur becomes feeble or extinct, with dulness on percussion, especially in the inferior portions of the lungs. The physical signs resemble very much those of hydrothorax, but there is, of course, no egophony, and there is less extensive dulness on percussion. Evidences of pulmonary œdema seldom occur until towards the close of the disease; indeed, its existence is often only noticed in the post-mortem examination of the lungs. In the form of an effusion into the air-cells, however, it is sometimes sudden and dangerous. I have known it fatal in a few minutes after it had produced alarming dyspnœa, and with the serum even gushing from the mouth of the patient.

Serous effusion may also occur in the brain, either in the pia mater or in the ventricles. It is difficult to say whether the symptoms indicating cerebral pressure are the result of simple congestion of the vessels, or whether this has terminated in effusion. It is probable that the tendency to coma, often observed during the close of the disease, is the result, in part at least, of serous effusion.

Finally, you will sometimes find after death œdema of the cellular tissue of the alimentary canal; but there is no evidence that this induces any symptoms during life. Enlargement of the liver from congestion, which can often be felt during life extending some inches below the ribs, is of common occurrence

in the advanced stage of the disease; and this passive congestion sometimes enlarges the spleen also.

The accumulation of dropsical fluid in the cellular tissue of the trunk, of the upper extremities, and especially in that of the face, in the cellular and vesicular tissues of the lung, in the submucous cellular tissue of the intestinal canal, as noticed after death, are by no means of unfrequent occurrence, but evidently hold a subordinate place to the effusions first noticed. You will seldom see, unless in very bad cases, that general, and often enormous bloating of the whole cellular tissue, that you will so frequently notice in the dropsy of Bright's disease.

The influence of the dropsical effusion in aggravating the symptoms of the disease is often painful to contemplate. At first, the gradual swelling of the lower extremities creates only a sense of uneasy fulness; but as the skin, stretched to the utmost, becomes tense and shining, it becomes a source of much distress. Inflaming from the slightest irritation, an erysipelatous redness and heat ensue, and even extensive superficial gangrene may follow. I have known even the scrotum to slough, and the testicles to be exposed by the fearful ravages of this complication.

A painter, aged 31 years, entered this Hospital with the signs of simple enlargement of the heart, the apex striking between the sixth and the seventh ribs, and outside the nipple. No morbid sounds existed over the heart. The impulse was increased. The patient had been affected with symptoms resembling asthma for a year past. He had a slight cough, and frequent attacks of dyspnœa, occurring principally at night, which, after lasting from eight to ten hours, were usually relieved by a free expectoration. Since his admission, swelling of the legs began, which soon became considerable, and the physical signs of hydrothorax occurred on both sides of the chest. After the patient had been a month in the Hospital, and had somewhat improved in his general condition, gangrene attacked the skin of the left leg, and in the short period of a single day it occupied nearly the whole calve, and a portion of the inside of the thigh. In the neighborhood of the knee, small, elevated, bluish lines were traceable, not caused by the veins, but apparently by a thinning of the skin, with serum

beneath. The inside of the right thigh also was colder than the surrounding parts, and presented the same bluish lines, although very faintly. In two days the gangrene had extended as far as the groin of the left extremity, where a dark line marked its termination. A large quantity of turbid serum exuded from the whole gangrenous surface. The patient did not long remain in this sad condition, but rapidly sank and expired.

The post-mortem examination verified the diagnosis of the case. The heart was much enlarged, but no valvular disease was discovered. A double hydrothorax existed.

More frequently, however, it happens that small and numerous papulæ appear on the distended skin, a little vesicle forming on their summit, which bursts, giving discharge to a constant oozing of serum; or the skin cracks superficially, and the serum oozes from the fissures: thus the distension is partially relieved.

But another serious difficulty is experienced when the abdomen, and especially when the chest, become the seat of considerable effusion. The dyspnœa, which could before be kept somewhat under control by rest, now becomes a source of constant, and often of inexpressible, suffering. The patient, bolstered up nearly in a sitting position in bed, dozes a little in unquiet slumber, breathing hard and heavy, but is soon aroused by an intolerable feeling of distress and sinking, and seeks for comfort in his chair. But no relief comes. Thus long and weary nights are passed, and days of constant distress, until at length exhausted nature gives up the struggle. Death sometimes ensues suddenly—sometimes after an interval of insensibility.

The digestive organs generally continue their functions without material disturbance during the early stage of enlargement of the heart, and if they fail at a more advanced period, this seems to accompany the general failure of the vital powers. There is one symptom, however, of disordered digestion, which I have found so constantly connected with this disease, and often existing alone, that I am disposed to regard it as something peculiar —this is flatulence. It is seldom absent in confirmed cases, and is a very unmanageable, and often distressing symptom, by the pressure it produces upon the diaphragm, and consequently upon

the heart and lungs. Severe paroxysms of dyspnœa are often induced by this cause, and even symptoms of angina pectoris, which are at once relieved by free eructations.

The condition of the pulse has long occupied the attention of physicians in the diagnosis of enlargement of the heart. I believe, however, that it possesses but little real value as a diagnostic symptom. I have generally observed that the pulse was more or less accelerated in these cases; sometimes, however, it preserves the natural standard, or it may be even more slow than is natural. But every variety, as to fulness, force, and regularity, exists in cases apparently similar in their anatomical changes, and even in the same case at different times. Take a simple case—enlargement of the left ventricle. In the early stage you may expect to find the pulse strong and full, but as the disease advances it may become feeble and small, and yet the heart may have increased in size during the period that has elapsed. So it may continue regular throughout the disease, or finally become irregular, as the energy of the heart fails. The same is true in other forms of enlargement—the pulse is a very uncertain guide as to its extent or its seat. Even the full and strong pulse noticed in connection with enlargement of the left ventricle, often occurs without such enlargement, but as a symptom of general plethora, or of some remote irritation. An irregular pulse has also been regarded as indicative of heart disease, and especially of valvular disease. It is true that if you find the pulse irregular, your attention would naturally be drawn to the condition of the heart, and you will often find it diseased. Sometimes it is simply enlarged; sometimes this is complicated with valvular disease. It is in the more advanced stage of enlargement of the heart that this irregularity is noticed. It may occur, however, as already stated when speaking of the action of the heart itself, without disease of that organ—in nervous subjects, when excited or irritated; in debilitating diseases, as typhus fever, and finally, in some persons as a constitutional peculiarity, without any apparent cause.

LECTURE XXX.

VALVULAR DISEASE OF THE HEART.

Organic changes in the valves from inflammation, from atheroma.—Physical sign of valvular disease—the endocardial, or blowing sound: its modifications in connection with the different orifices of the heart.—Illustrative cases.—Cyanosis.

In the two preceding lectures, I have occupied your attention with the pathology and symptoms of enlargement of the heart. I am far from having finished all I wish to say on this important subject. But I will leave it for the present, to speak of another subject, valvular disease of the heart.

Valvular disease is usually treated in the books, as something quite distinct from enlargement of the heart. But, clinically considered, they are so intimately connected, that the one may be regarded as a part, as a complication of the other. My reasons for taking this view of the subject are these. Valvular disease presents no rational or constitutional symptoms by which it can be distinguished from simple enlargement of the heart. When it exists as the primary lesion, it is so soon followed by enlargement, that the latter may be regarded as the principal affection. Finally, the evidences of valvular disease may be wanting, even when it exists, and that at a time when those of enlargement of the heart are most distinct. The truth is, the signs of valvular disease are purely physical, and are so far modified by remote and by occasional causes, that they may exist or not, with the same degree and kind of organic changes in the heart.

Let me first call your attention to the morbid changes produced by disease in the valves and in the orifices of the heart; for the two must be carefully studied together, although, for the sake of brevity, I commonly use the term valvular disease as applicable to both. Although, in a vast majority of cases, it is the disease of the valves that affects the orifices, yet, in strict observation, it may happen that the orifices are alone affected,

the valves remaining perfectly natural; as, for instance, when an orifice is simply dilated and the valves connected with it preserve their natural condition. Yet even in this case, in a clinical point of view, it may be said that the valves are in fault, for they cannot close the orifice to which they belong; so that precisely the same impediment to the circulation, and the same physical signs will occur, as if the valves themselves were shrivelled, or wasted by disease—the orifice preserving its natural condition. So that, after all, when I speak of valvular disease, as applied to the morbid condition both of the valves and of the orifices, I am using language which is practically, if not anatomically and strictly, correct.

The valves of the heart are, as you well know, formed by the folding of the lining membrane of the heart upon itself. These folds are strengthened by the interposition of a layer of fibrous tissue, which, in the auriculo-ventricular valves, may be regarded as an expansion of the chordæ tendineæ. It will thus be seen that the textures which enter into their composition are very analogous at least, if not identical with those which enter into the composition of the joints. You can then easily understand why a rheumatic inflammation of the joints, especially if general and attended with much constitutional disturbance, may affect also the valves of the heart by metastasis, or by coincidence. I have stated the fact, that these results, especially the latter, often really happen, when speaking of the causes of enlargement of the heart. I repeat, now, that acute articular rheumatism frequently attacks the valves of the heart, either openly or insidiously, and thus lays the foundation, first of valvular disease, to be, more or less speedily, followed by enlargement of the heart. I say that it attacks the valves, because the valves are the primary, and often the only seat of the attack, and it is in them that the chief danger resides. But the lining membrane of the heart may be similarly affected, leading, if the inflammation be extensive, to a rapidly fatal result; while, if limited in extent, it appears to exert but little influence, either in the acute or chronic progress of the case.

Acute rheumatic inflammation, when it attacks the valves of the heart, produces swelling, redness, and softening of the tis-

sues. Usually, you will find an exudation of lymph upon the free surfaces, and there is no doubt that a similar deposit takes place in the very delicate cellular tissue that unites the external membrane to the intermediate fibrous layer. In time, the redness, softening, and, in a great degree, the swelling disappear, but the real mischief is but just commencing. The lymph effused in the cellular tissue becomes organized and contracts, and thus the valve becomes shrivelled, rigid, opake, while it remains permanently thickened. This increased thickness, however, is not accompanied by increased strength in the valve; it becomes more friable, more easily torn by the pressure of the blood.

The lymph, also, that has adhered to the free surface of the internal membrane, becomes organized, and contracting in its turn, increases the rigidity, the shrivelling, the thickening of the tissues. Sometimes it first unites the free edges of the valves, and then, contraction taking place, the corresponding orifice is much diminished in size. Sometimes, rarely, however, a portion of the valve is bent backward against the lining membrane of the heart and firmly united to it, so as to become entirely useless in function. The effused lymph appears upon the valves in the form of patches, or, more frequently, as vegetations of a rosy hue; sometimes smooth and globular, but usually irregular in shape, clustered, and with the surface subdivided by fissures—resembling, in fact, the venereal vegetations which appear upon the penis and about the anus. These patches and vegetations may assume, in time, a cartilage-like, or even bone-like condition. The lining membrane of the heart itself, if the inflammatory action has extended to it, may present the same morbid conditions—shrivelling, thickening, opacity, vegetations.

The chordæ tendineæ of the valves, and even the columnæ carneæ, which belong properly to the muscular structure of the heart, are sometimes found shortened, contracted, so as no longer to allow the corresponding valve, or a portion of it, to rise to its proper level, and thus close the orifice of the heart. This change is probably the result of inflammatory action, and analogous to the thickening and shrivelling of the valves from the same cause.

I may state the important fact, that valvular disease is much more frequently found in the left than in the right side of the heart.* Indeed, the little knowledge we possess of disease of

* The recently published observations of Dr. Blackiston, of Birmingham, England, seem to show that disease of the right side of the heart is much more frequent than other observers have supposed it to be. I have stated, in speaking of enlargement of the heart, the comparative frequency with which he found the right and left ventricles affected with that form of disease. The same difference from other observers is noticed in his statistics of valvular disease. Thus, he found in his 156 post-mortem examinations,

Aortic orifice incomplete	39	times.
Pulmonary orifice incomplete	5	"
Mitral orifice incomplete	67	"
Tricuspid orifice incomplete	111	"
Tricuspid obstruction	1	"

The author does not mention the kind of disease that induced the imperfect condition of the orifices. He states, however, that sixty of the cases of tricuspid regurgitation were the result of simple dilatation of the orifice, the valve not having increased proportionately in size so as to close the orifice. This accident has generally been regarded as connected with the dilatation of the right ventricle during the last few days of life, and has hardly been looked upon as a diseased condition. According to our author, however, the sign of this condition, viz., swelling and pulsation of the jugular veins, often occurs long before death, generally preceding, in fact, the dropsical effusion. Indeed, he is disposed to regard this effusion as chiefly caused by the obstruction to the circulation produced by tricuspid regurgitation. While the general opinion (and with this I am disposed to agree) is, that the obstruction to the circulation most frequently arises in the left side of the heart, or even in some remote part of the system; and that both dropsy and tricuspid regurgitation are the result of this obstruction, not the cause of it. In the analysis of forty-eight cases, made some years ago, I arrived at the following results:

Traces of Endocarditis, including Ossification.

Aortic valves	25	times.
Pulmonary valves	3	"
Ossification of valves of the right side of the heart	1	"
Mitral valve	29	"
Tricuspid valve	20	"
Ossification of valves of the left side of the heart	13	"

Bizot's examinations give still more decided results in favor of the right side of the heart.

Aortic valves thickened and opake	45	times in	157	cases.
Pulmonary valves thickened and opake	9	"	157	"
Mitral valve with opake spots and ossification	24	"	156	"
Tricuspid valve diseased	2	"	156	"

The researches of Dr. Taylor tend to prove that endocarditis, and the changes it induces in the valves, are quite as frequently associated with Bright's disease as

the valves of the right side is derived from post-mortem examinations. For even when important changes in these valves have taken place, the action of the right side of the heart is usually too feeble to develop any physical sign during life.

It is easy to perceive the physical effects of these changes in the valves: obstruction of the orifices of the heart interrupting the flow of the blood onward, imperfect closure of the orifices admitting the flow of the blood backward.

Experience teaches that a large proportion of the cases of valvular disease occurring in children and in adults who have not reached the middle period of life, are rheumatic in their origin. After the middle period of life, the rheumatic cause becomes less frequent, but the number of cases of valvular disease does not diminish. In fact, a new cause of valvular disease makes its appearance at this time, and continues to increase in frequency as age advances. This is the atheromatous deposit in the valves. I would not be thought to say, that the rheumatic cause exists only in early life, or that the atheromatous deposit is only found in those who have passed the middle period of life. Excep-

with rheumatism. Thus, according to his statement, acute endocarditis occurs in 7 per cent. of the cases of acute rheumatism, and in 8 per cent of the cases of Bright's disease. Again, the same author states, that in 75 cases of acute rheumatism, valvular disease, old or recent, existed in 34 cases; while in 50 cases of Bright's disease, valvular disease, old or recent, existed in 25 cases. Thus, if these statements can be depended upon, and they appear to have been prepared with great care, Bright's disease is quite as frequent a cause of valvular disease as rheumatism. Again, as Bright's disease usually terminates fatally at a comparatively early period of life, 50 in 74 cases, reported by Dr. Bright himself, terminating before the age of 45 years, and 27 in 33 cases, reported by Dr. Malmsten, dying as early as the age of 40 years, we should be led to expect valvular disease and its consequences, among the young, or among those who have not yet reached the middle period of life, in connection with Bright's disease quite as frequently as in connection with rheumatism. But do facts prove this to be true? While we have long been in the habit of tracing valvular disease to rheumatism, no author, to my knowledge, has before shown its connection with Bright's disease. I must confess, so far as my own experience extends, that while the tracing the connection of valvular disease with acute rheumatism is an every-day occurrence, I have never yet suspected Bright's disease as occupying any such position. Still, cases of valvular disease certainly occur in early life that cannot be traced to a rheumatic cause, and as Bright's disease is often obscure in its symptoms, its connection with such cases may have been overlooked. The subject is well worthy a more extended examination.

tional cases may occasionally be met with; but the general rule will be found to be as I have stated it. The deposit of atheroma has long puzzled pathologists as to its true nature and seat. Every one has noticed it who is at all conversant with examinations of the body after death, for it is found in the aorta, or upon the valves in almost every person advanced in life. Recent and improved examination has discovered that this deposit contains abundant globules of fat, with crystals of cholesterine, and some earthy matter. Its seat is in the cellular tissue which connects the external membrane of the valves to the intermediate fibrous tissue. It is infinitely more common in the valves of the left side of the heart than in those of the right side. It attacks that portion of the valves called the adherent edge, in contradistinction to the vegetations, which prefer the free edge of the valves. In the mitral valve, it is seen most distinctly upon the face which looks towards the ventricle. In the aortic valves, on the contrary, it is usually noticed first upon the face that looks towards the aorta, and in both cases elevating very slightly the delicate and transparent external membrane of the valve, beneath which it is situated. These deposits, at first small, grow larger and coalesce, and in their early stage seem simply to render the valve more thick and clumsy than natural, although sometimes a thick ring of them will form around the base of a valve, and thus produce some obstruction of the corresponding orifice. But in time more important changes ensue in the valve affected. These atheromatous deposits soften, the external membrane of the valve is destroyed, as well as a portion of the intermediate fibrous tissue, and thus the valve loses a portion of its substance. In other cases, the atheromatous deposits, instead of softening, become changed into a calcareous matter, thus constituting the most frequent cause of what is called ossification of the valves. These bone-like deposits, the external membrane of the valve having been removed by absorption, often lie directly in contact with the current of the blood, adhering, sometimes firmly, sometimes loosely, to the surrounding tissues. Occupying a considerable portion of a valve, they render it rigid; projecting into the corresponding orifice, they obstruct it; deforming the valve, they destroy its power of closure.

It is as easy to perceive in this form of disease, as in the rheumatic form before described, what the consequences may be: obstruction to the flow of the blood onward, permission to the flow of blood backward.

The physical consequences of valvular disease, then, may be briefly stated to be obstruction and regurgitation. Either may exist alone, or both may be united in the same valve, or each may occur from disease in different valves, but at the same time. The general and uniform result of either of these conditions, is an impediment to the circulation of the blood through the heart, leading to enlargement of that organ.

There are two points in connection with the subject of valvular disease which I wish now to notice, because I think that they throw light on some otherwise obscure points of diagnosis. It has happened to many observers to notice the physical signs of valvular disease during life, and after death to find the valves and the orifices in an apparently natural condition. Thus, they have been led to believe that but little reliance could be placed on these signs. But I believe that in these cases the *naturalness* is but apparent, not real. Suppose that a valve retains its natural appearance, especially its natural size, and the corresponding orifice enlarges with the general enlargement of the heart? The time may come when the valve will no longer be able to close the orifice, and regurgitation will ensue. Yet, unless the size of the orifice be measured and carefully examined, no apparent fault will be found to exist; or, on the other hand, the portion of the heart affected may gradually enlarge by hypertrophy of its walls, and by the dilatation of its cavity, and yet the corresponding orifices may not dilate at all. In this case, especially if the heart is capable of vigorous action, a virtual obstruction of the orifices exists, although they may appear natural on a superficial examination after death. A want of due attention to these circumstances has led some excellent observers to doubt the value of the signs of valvular disease.

The existence of valvular disease of the heart is revealed by a single physical sign—*a blowing sound*, heard chiefly over the region of the valves, and which has been properly designated as the endocardial sound. I have already, in a former lecture, en-

tered fully into an account of the mechanism of this endocardial sound, so that I need now only occupy myself with its diagnostic value, as indicating the different forms and seats of valvular disease. If it were enough to apply the ear to the precordial region, and recognize a murmur or unnatural sound there, to establish the existence of valvular disease, you would, by this simple process, have ascertained the chief object of your investigation, since it is of far more importance to establish the simple fact of valvular disease than its precise seat or character. But the real difficulty is much greater. You may, in fact, hear a blowing sound over the valves, which is not at all connected with valvular disease, but in which the heart, so far as its structure is concerned, is quite healthy. This happens in chlorosis, in cases in which anemia has been the result of hemorrhage. I wish only to allude to this fact at the present time. I can better point out its diagnostic value on another occasion. Again: you have the friction sound which occurs in pericarditis, and which sometimes so closely resembles the blowing sound of valvular disease, that the most careful ear cannot always distinguish the difference. The diagnostic value of this friction sound has been fully pointed out when I considered the physical signs of pericarditis. The point I wish now to impress upon your minds is this—that a blowing sound over the heart does not necessarily indicate any valvular disease whatever, and also, that, while it appears to be internal, or endocardial, it may, in fact, be external, or pericardial.

Suppose that the sound heard over the heart is connected with valvular disease, does it possess a uniform character? By no means. I have used the term blowing, because this is the most frequent, the earliest, the simplest character of the sound. It is often the only sound you can hear during the whole progress of the disease; and in cases in which it becomes changed, it often exists in the early stage, and appears and reappears, perhaps many times during its progress. The truth is, the sawing, rasping, filing, and even the musical sounds heard in cases of valvular disease, are all modifications of the simple blowing sound, and may all be united by the term endocardial sound. A simple blowing murmur, as I have stated in a previous lec-

ture, becomes raised in its key, and more harsh, by an increase in the obstruction of the orifice at which the sound is generated, or by its becoming more rough or rigid. The same result may be produced by whatever adds to the force of the heart's action. Thus the gradual progress of valvular disease, by which the affected orifice is rendered more narrow, or more rigid, or more rough, will change the soft blowing sound to one that is harsh and rough. Again: without increase of valvular disease, the simple enlargement of the heart, by increasing its action, may lead to the same result. Both these causes of change in the valvular sound are operating in many cases, so that it is no wonder that the soft blowing sound of the early disease shall have become changed into a harsh, or sawing sound, when the disease is fully developed—when the condition of the valves and of the muscular power both unite in its production.

But there may be a limit to these influences, especially to that of the heart's action in producing a harsh or rough endocardial sound. The power of the heart fails at length, although its enlargement may increase. This is a fact I have already sufficiently considered in another lecture. With this failure of the vital power of the heart, the endocardial sound may grow more soft and blowing, notwithstanding that the valvular disease is unaltered, or is even increasing. It may cease altogether, as the feebleness of the heart's action increases. The exact influence which the action of the heart itself, on the one hand, and the condition of the valves on the other hand, can hardly be appreciated. When the orifice affected by valvular disease is very rough and rigid, and the contraction not very considerable, a feeble heart may generate a murmur, and even a rough sound. So, when the orifice is but little contracted and is comparatively smooth, a strong heart may generate a strongly marked sound.

As a general rule, the changes in the valves and in the corresponding orifices being gradual, the physical signs connected with them are gradually developed. But this is not always the case. A striking case has recently occurred to me, in which the aortic valves, weakened by inflammatory changes in their structure, during the convalescence from an attack of fever, suddenly gave

way, so as almost to destroy entirely the action of these valves. Regurgitation was almost as great as if no valves had existed. In this case no sound was developed; the regurgitating orifice was too large for that. But there was another reason. The heart was overpowered by the congestion of the left cavities, for the quantity of blood that flowed back into the left ventricle during the diastole must have been nearly as great as that expelled by the previous systole. Examination disclosed a very rapid and tumultuous action of the heart, a confused, rushing sound in place of the natural sounds, while the pulse retained considerable strength, and was regular in its action. The dyspnœa was very marked, and the patient soon expired. This sudden rupture of a valve, but in a less degree, is probably not a very rare occurrence, and then the signs of regurgitation might suddenly ensue. I think I have known this happen even when no evident previous disease existed, as after a sudden effort, in which a portion of a valve, or perhaps a tendon connected with a valve, had suddenly been ruptured, and a regurgitating murmur, with symptoms of disturbed circulation, ensued. I recollect very well the case of a child, about eight years of age, who was suddenly seized with symptoms of heart disease, after attempting to carry some heavy books up stairs. In this case I noticed a murmur indicating mitral regurgitation, which continued more than a year, and finally ceased.

A blowing sound, often rendered rough, sawing, rasping, musical, by the circumstances already mentioned, is the leading characteristic of the endocardial sound—the indicator of valvular disease in many cases, but not always. Let me now advance a step further, and discover, if I can, whether there are any facts which connect this sound with valvular disease with more certainty. Suppose that you listen for this sound, day after day, and always hear it, especially under circumstances of unusual quiet? Suppose that you always notice a degree of roughness in the sound, even when the action of the heart is not much increased? What are the indications from these facts? They certainly point to valvular disease. Permanency of the sound is undoubtedly an indication of valvular disease, although not a certain indication; for the valvular disease may be so slight as

not to generate any sound, when the action of the heart has been quieted by rest, or by other means. Roughness of sound is also generally an indication of valvular disease, although not a certain indication; for a very violent action of the heart, in chlorosis, or in anemia, may cause a degree of roughness in the sound heard over the heart, and yet no valvular disease be present. The inference from these facts is plain. A rough sound, with moderate action of the heart, is a strong indication of valvular disease. A permanent sound, with a moderate action of the heart, is also a strong indication of valvular disease.

The permanence or the roughness of the endocardial sound, especially in connection with moderate force of the heart's action, establishes the probability of valvular disease; but you can often attain to a much greater degree of precision. You can, in many cases, establish the precise seat of the endocardial sound, and ascertain whether it be connected with the flow of the blood onward, or with regurgitation. Now every endocardial sound connected with regurgitation is, probably, organic, that is, connected with changes of structure, and so are those connected with the flow of the blood onward, *with one exception*. The endocardial sound connected with obstruction of the aortic orifice, may be imitated by an endocardial sound having no obstruction as its cause. It may be an inorganic sound. The whole difficulty, then, in distinguishing between organic and inorganic endocardial sounds is reduced to this narrow point—to distinguish between the sound indicating obstruction of the aortic orifice, and that induced by chlorosis, or anemia. Let me now inquire what are the signs of obstruction of the aortic orifice, and how you can distinguish them from those produced by inorganic causes?

Obstruction of the aortic orifice is indicated by an endocardial sound, having its maximum over the aortic valves: it accompanies or masks the first sound of the heart: it is transmitted, by the current of the blood, upward, in the course of the aorta.

In cases of chlorosis and of anemia, you will hear the same sound. But it is usually soft and blowing, it is intermittent, and especially it is unattended by any of the signs of enlarge-

ment of the heart. It is also frequently heard in arteries too remote from the aortic orifice to be transmitted from that source. as in the carotids, or even in the femoral arteries.

Imperfect closure of the aortic orifice is indicated by an endocardial sound, having its maximum over the aortic valves: it accompanies or masks the second sound of the heart. It is transmitted, with the current, in the direction of the left ventricle.

This endocardial sound is a sound of regurgitation, and implies organic disease. It is usually softer and shorter in its duration than that connected with aortic obstruction. It is seldom transmitted more than an inch or two over the body of the left ventricle, although I have heard it as far as the apex of the heart.

I will relate cases which will serve to illustrate the most important facts in the diagnosis of aortic obstruction and regurgitation. In these cases, the physical signs of enlargement of the heart are also worthy of your attention.

A negro porter, aged forty-nine years, of short stature and stout frame, had an attack of acute rheumatic fever ten years ago, which did not cease entirely until six months had elapsed. His health, after this attack, continued good until about eight months since, when he began to notice a stricture across the chest, attended by dyspnœa. About a month afterwards he noticed palpitations, and a cough ensued. Two months ago he began to notice a swelling of the feet, which gradually extended up to the abdomen. When I saw him, a month after this time, he presented all the symptoms of enlargement of the heart and of valvular disease, with anasarca and ascites. He had also a fatiguing cough, with mucous expectoration streaked with blood, and attended with much dyspnœa. Auscultation discovered an abundant mucous rattle over the postero-inferior portions of both lungs, with the sibilant and sonorous rhonchi in the other portions of the chest. The liver was enlarged, extending as low as the umbilicus, and was tender on pressure. The pulse was full, strong, and regular, with a slight rebound.

The patient was bled freely from the arm, and purged; and these remedies were followed by a pill of calomel, squills, and

digitalis, but with only partial relief. He soon became worse, and could not lie down; his head was also hot, and he had lost the power of speech, with a tendency to restlessness and delirium. His right arm was paralyzed, but without loss of sensibility. At one time he complained of sudden and severe pain in the head. The pulse was one hundred in a minute, full and strong. He was again bled freely and purged with Croton oil, and with great relief. The anasarca subsided very considerably, the breathing became easier, so that he could lie nearly in a recumbent position, and could sleep quietly. The liver had much diminished in size. The pulse fell to seventy-eight, and became soft and more weak.

The heart, after repeated examinations, presented, uniformly, the following physical signs:

Dulness over the precordia increased; absence of the respiratory murmur in the precordial region. The apex strikes one inch and a quarter below and outside the left nipple. Over the aortic valves, the first sound of the heart is a little rough. The second sound, in the same region, is much more rough, and is prolonged. It has its maximum of intensity over these valves, that is, at the centre of the sternum, between the cartilages of the third and the fourth ribs. It extends, however, up the aorta, and nearly over the whole heart, where it is most diffused in its character. The action of the heart is moderate, and at times a little irregular.

Diagnosis—Enlargement of the heart, imperfect closure of the aortic orifice, with slight roughness of the same orifice; general bronchitis; congestion of the liver; cerebral disease.

In the progress of the case, the intelligence was much impaired, and the power of speech continued to be lost. The right leg became paralyzed. The patient became affected by sudden attacks of unconsciousness, attended by screaming, which would subside into a slight stupor. The heart symptoms continued unaltered. Purging with Croton oil relieved him. The pill of calomel, squills, and digitalis, carried to slight salivation, did no good. The patient was removed to the Retreat, at Staten Island, where he died in a few days, ten months after the attack, and about two months after he came under my observation.

I did not attend the post-mortem examination, but had sent my diagnosis of the case to Dr. Boardman, at that time the physician to the Retreat, who informed me that it was confirmed in every particular. The heart was enlarged, chiefly in the right ventricle, which was much hypertrophied and somewhat dilated. The left ventricle was natural. The aortic valves were small, and unable to close the orifice, but not diseased in structure. The aorta was dilated.

It is to be regretted that the brain was not examined in this case. It is probable that there was congestion of this organ, and apoplectic softening in the left hemisphere.

Notwithstanding the existence of aortic regurgitation, the pulse was full and strong, until reduced by blood-letting. It had the slight rebound which is sometimes noticed in such cases. The right side of the heart was principally affected; and it will be remembered that dyspnœa was the first symptom in the case, and that dropsical effusion occurred early. This tends to confirm the opinion entertained by some observers, that affections of the right side of the heart are most apt to induce such symptoms. How far the rheumatic attack may have been concerned in producing the affection of the heart, is not apparent.

It will be noticed also in this case, that although the enlargement of the heart did not extend to the left ventricle, yet there was a very considerable displacement of the apex; also, that the regurgitating sound was rough and much diffused over the heart, which, I think, rarely happens. Yet, it appears to have been a case in which regurgitation existed in its most simple form, from a want of sufficient size of the valves. The left ventricle, however, was not diseased, and the action of the heart was of good strength. This, probably, had more influence in rendering the sound rough and extensive than the condition of the valves.

A mason, aged thirty-three years, of a moderately robust constitution, and a free liver, had always enjoyed good health until about ten weeks since, when he was attacked with what he considered a common cold. About six weeks before I saw him, he was first affected with palpitation, and by dyspnœa, with inability to lie down. The dyspnœa continued to increase until three weeks ago, when it became excessive, and was relieved by a pill

of Croton oil, which brought away, according to his statement, three pails full of watery discharge from the bowels. About a week since, he began to notice some swelling of the feet.

The countenance is pale and morbid; the lips are blue; there is cough, with a mucous rattle posteriorly, in the chest.

The apex of the heart strikes two inches below, and one inch outside the left nipple; there is a rough, blowing sound with the second sound of the heart, extending along the aorta, receding over the heart; the pulse is regular, soft, with a slight rebound or back stroke.

Diagnosis—Enlargement of the heart; aortic regurgitation.

Eight days after this examination, the patient was in articulo mortis. There was orthopnœa; the countenance was pale; the intellect was not affected; the skin was cold; the pulse feeble; and slight œdema of the feet existed.

On post-mortem examination, the pericardium contained about one ounce of serum; and white patches of old lymph were noticed on the surface of the ventricles; the heart was generally enlarged, the left ventricle more than the other portions; all the valves were healthy, except the aortic valves; the free edge of one of these valves presented a distinct loss of substance, an indentation with bevelled edge, and evidently of old date; another of the valves was nearly half destroyed, and with ragged, broken edges, evidently a recent destruction.

The lungs, especially the right, were connected to the ribs by old adhesions; about a pint of serum was found in the left pleural cavity; the lungs were congested, and the bronchi reddened; the liver and the mesenteric veins were congested; the spleen was natural.

Obstruction of the mitral orifice may be indicated by an endocardial sound, having its maximum over the mitral valve; it may accompany or mask the second sound of the heart; it may be transmitted, with the current of the blood, in the direction of the left ventricle.

I have never heard this endocardial sound in a single instance, although I have not unfrequently examined cases in which a marked contraction of the mitral orifice existed. Dr. Hope has been more fortunate; and I make the statement I have just

made on his authority. It is confirmed also by analogical reasoning. This murmur, if it exists at all, is probably always very soft and feeble.

I will relate a case as illustrative of this form of disease, and I might easily mention many analogous cases.

A lady had been ill for two years when I was first called to attend her. She was first attacked with severe dyspnœa, which had continued to recur in paroxysms, although she had never since been entirely free from it. For some time she had been subject to palpitation; but it had never been distressing. She had not been subject to rheumatism.

At the time I first saw her, she was suffering from an attack of active congestion of the left lung, indicated by a coarse crepitation at the base posteriorly, and extending into the lateral region of the chest, where slight dulness on percussion also existed. The pulse was somewhat accelerated, regular and soft.

The heart presented the physical signs of general enlargement. There was increased dulness over the precordial region; the apex struck further to the left than natural, and between the sixth and seventh ribs. The action of the heart was moderate. The impulse was stronger to the right of the sternum than to the left; but strongest at the ensiform cartilage. No morbid sounds existed over the heart.

She was ordered a blister to the left side, the tincture of hyoscyamus with the camphor mixture and a good diet, as she was feeble. During this treatment she somewhat improved, and could lie down and sleep comfortably. But after the interval of a week, she was seized with great dyspnœa almost amounting to asphyxia; orthopnœa; lips livid, pulse very feeble, skin cool. The physical signs of pneumonia in the second stage existed at the base of the left lung. The action of the heart continued regular and superficial. No morbid sounds existed.

On post-mortem examination, between one and two pints of transparent serum was found in the left pleural cavity. The lower half of the left lung was congested and heavy, and presented traces of lobular and vesicular pneumonia. The bronchial tubes, at the root of the lung, were congested, and contained some opake mucus. The pericardial sac was healthy, but

the heart was considerably enlarged, and entirely at the expense of the right side. The left ventricle was small. The mitral valve was diseased; its free edges were adherent, thickened, and contracted, but smooth. A few vegetations were noticed on the auricular surface; and the lining membrane of the left auricle was thickened and opake. The mitral opening was so small that it would not admit the end of the little finger. The valve appeared capable of closing the orifice, so that no evidence of regurgitation existed. The pulmonary orifice was dilated, but the pulmonary valves were increased in proportion, both in size and thickness. The coats of the pulmonary artery were quite as thick as those of the aorta.

In this case you will notice that enlargement of the right side of the heart existed, with dyspnœa as the first and most prominent symptom; yet the first step in the morbid series was, probably, on the left side of the heart—viz., the contracted state of the mitral orifice. The obstruction to the circulation was backward from this point, through the pulmonary vessels, and the enlargement of the right ventricle and of the pulmonary artery was the direct consequence. The affection of the mitral valve was evidently inflammatory, yet the patient stated that she had never suffered from rheumatism. This is one of the cases in which pulmonary apoplexy is most apt to occur. Indeed, you cannot have two more powerful causes of this accident than in the obstruction of the mitral orifice preventing the free return of blood from the lungs, on the one hand, and on the other hand, the increased development of the right ventricle with a free condition of the orifice of the pulmonary artery, constantly forcing the blood into the lungs; yet in this case it did not occur.

There was no blowing sound heard in this case, and this corresponds with my uniform experience in similar cases of mitral obstruction. It should be observed, also, that the action of the heart was feeble. The effect of mitral obstruction in rendering the left ventricle smaller even than natural, is worthy of notice; also, as an evidence that pulmonary obstruction, that hydrothorax was the only form of dropsical effusion.

The influence of this hydrothorax occurring on the same side in which a slight pneumonia existed, was chiefly to increase the

distinctness of the physical signs of the latter disease, namely, the dulness, and the intensity of the bronchial respiration. It is probable that a broncho-egophony existed which might have led to the detection of the liquid effusion, but no mention is made of this fact. This effect of a moderate effusion in heightening the signs of pneumonia, accords with my observation in many cases.

Finally, the case was one of those cases which, without auscultation, might readily be mistaken for an asthmatic affection of the lungs. Yet the disease of the lungs was evidently recent, while the dyspnœa, with paroxysms, had existed for two years.

Imperfect closure of the mitral orifice is indicated by an endocardial sound, having its maximum over the apex of the heart: it accompanies or masks the first sound of the heart; it is transmitted, not upward with the current, but downward along the chordæ tendineæ, and is heard most distinctly near the apex of the heart.

This endocardial sound is perhaps the most important of all the endocardial sounds, from its frequent occurrence and its distinctness. Its mode of distribution is different from that noticed in other forms of valvular disease, but the reason for this will be apparent after a moment's reflection. It is not transmitted upward with the current, because the walls of the left auricle are, at the moment of its occurrence, relaxed, and incapable of transmitting sound. All the vibrations seem to be caught, so to speak, by the tense chordæ tendineæ, and carried towards the apex, where these chords, by the intervention of the columnæ carneæ, mostly terminate. It is this endocardial sound, heard most distinctly at the apex, but diffused sometimes over the body of the left ventricle, which is most easily mistaken for the pericardial friction sound, which may have the same seat. But this friction sound has one striking characteristic, if you will but wait for it. It is of short duration, while the endocardial, or blowing sound, is usually permanent. The former disappears as soon as adhesion takes place; the latter is most frequently connected with valvular disease which it is exceedingly difficult to remove. The endocardial sound is, however,

sometimes intermittent, ceasing for a time, when the action of the heart is feeble. Again, a friction sound is apt to change its place from time to time, moving a little in different directions. Finally, I must caution you against placing too much reliance upon the mere character of the sound. With all the attention I have been able to bestow, I have sometimes mistaken the blowing sound for a friction sound, at the first examination.

I will mention a case in which this important form of valvular disease, mitral regurgitation, existed in connection with some enlargement of the heart.

A young gentleman, 23 years of age, had been subject, for eight or nine years, to heart symptoms; to palpitation, and especially to dyspnœa. Indeed, he told me that he thought his breathing had been rather less free than natural from early childhood. He had never suffered from rheumatism. For the year past, his cardiac symptoms have been growing more troublesome. Lately, his feet have begun to swell, and he is now confined to his room. When I first saw him, a few weeks before death, his chief complaint was dyspnœa. His countenance was pale, but not livid, he had not emaciated very much. His feet were œdematous, and there was evidently a serous effusion in the right pleural sac.

The heart did not present very decided evidences of enlargement. The apex, especially, was not displaced, and the dulness over the precordia was not materially increased. Afterwards, however, a distinct pulsation was noticed in the epigastrium, and referred to an enlargement of the right side of the heart. The action of the heart was irregular; the impulse was moderate; and a distinct, systolic, blowing sound existed at the apex. The pulse was very feeble, and irregular. The existence of mitral regurgitation was quite evident.

After a short time copious and repeated hæmoptysis occurred, which soon terminated the life of the patient, about three weeks after the note was made of his case.

Post-mortem examination disclosed a moderate enlargement of the right side of the heart, hypertrophy and dilatation, and of the left auricle. The left ventricle was quite natural in size. The lining membrane of the left auricle was much thickened,

and lined, first, by a thin layer of transparent lymph, and then by old and friable coagula, such as are found in aneurisms. The mitral orifice was reduced to an opening which would hardly admit the end of the forefinger. The edges of the valve seemed united by a sort of seam; the valve was not irregularly thickened, or the seat of vegetations. The regurgitation through this opening must have been considerable. The pulmonary artery was larger, and nearly as thick as the aorta. All the valves and orifices of the heart were capable of performing their functions except the mitral. A pint or more of turbid, reddish-yellow serum existed in each pleural sac, and there was also a slight effusion of lymph. The lower lobe of each lung was not compressed by the liquid effusion, but it was condensed, solidified, of a dark-red color, unaerated, a little softened, and discharged, on pressure, a large quantity of dark, thick, viscid blood.

In this case the mitral obstruction was quite as great as the mitral regurgitation, yet no physical sign of the former lesion existed at my examination. The pulmonary hemorrhage which occurred in this case, with an apoplectic condition of the lungs, is not unusual in this form of disease.

A boy, ten years of age, of delicate constitution, has been all his life subject to shortness of breath, and for the last four years to palpitations. During the last four months these symptoms have increased, and have occurred in paroxysms resembling angina pectoris. He is attacked by severe pain in the chest, in the precordial region, and extending down the arms, and sometimes down the legs, followed by numbness and tingling in the parts affected, and attended by a feeling of suffocation. He is also subject to attacks resembling syncope. His sleep is disturbed by startings and by bad dreams. His digestion is good, but he has emaciated somewhat. There is a trifling cough, but no dropsical symptoms.

On examining the precordial region, there is evident dilatation with increased dulness on percussion, measuring two inches and three-fourths by two inches; the respiratory murmur is absent. The apex strikes half an inch outside the left nipple. The action of the heart is strong, regular, and superficial. The first sound is masked over the left ventricle, by a rough, grating

sound, which softens into a blowing sound at a distance. This sound is, after repeated examinations, equally distinct at the base of the ventricle and at the apex; perhaps it is more intense at the base and more loud at the apex. The second sound is very clear and abrupt. The pulse is more feeble than the impulse of the heart, and accompanied by a thrill, and is one hundred and four in a minute.

This case progressed without any material change in the symptoms, or in the physical condition of the heart, for about six months, when it terminated fatally after a prolonged agony. Œdema of the feet commenced about a week before death. A post-mortem examination disclosed the following condition of the heart: pericardium healthy, containing about three ounces of serum; the heart was nearly double the natural size, from a general enlargement of the organ; the mitral valve was opake, and thickened to the extent of half an inch, the free edge being more particularly thickened and irregular, and with the under surface covered by vegetations. The left and posterior surface of the left auricle was also affected by vegetations.

There was considerable effusion into the pleural cavities, especially into the left cavity; the lungs were congested; the liver was much enlarged, and more firm than is natural; the spleen was congested, and filled with granulations.

I shall allude to but one fact in connection with this case, and that is a fact very frequently noticed in disease of the mitral valve. The pulse was decidedly more feeble than the impulse of the heart.

What I have said of valvular disease of the left side of the heart is true also, *mutatis mutandis*, of the right side. The maximum point of the endocardial sound, whether it accompanies or masks the first or the second sound of the heart, the direction with which it is transmitted must be carefully noticed, and the diagnosis established precisely in the same way as when the valves of the left side of the heart are affected. Still, we know really but little of valvular disease of the right side of the heart. It is comparatively rare, and when it does occur, the comparative feebleness of the action of this side of the heart would diminish the chances of the physical signs being devel-

oped, even if the organic changes which are calculated to produce them, really exist. The rules for the diagnosis, then, of valvular disease of the right side of the heart are derived from analogy, and from the few cases in which physical signs present themselves. Occasionally, a case of obstruction of the orifice of the pulmonary orifice has been observed. In this case, the endocardial sound has been found to be more superficial than in the case of aortic obstruction, and its transmission has been to the left, with an abrupt termination; along the pulmonary artery, to its bifurcation, instead of upward, along the course of the aorta. A case of imperfect closure of the pulmonary valves with regurgitation could not, I think, be distinguished from the same condition of the aortic valves, even if the endocardial sound existed, unless, indeed, it was most strikingly superficial.

If obstruction of the mitral orifice reveals itself but seldom by an endocardial sound, how much less frequently should you expect to find it in obstruction of the orifice of the tricuspid valve, since the action of the right side of the heart is naturally so much more feeble than that of the left side. The endocardial sound, indicating imperfect closure of the tricuspid valve, may possibly occur, but when it does not, you may notice another sign which indicates the same thing. This is the pulsation of the jugular veins, synchronous with the pulsation of the heart. This symptom, however, I think, often occurs towards the termination of heart disease, simply as the effect of a dilatation of the orifice beyond the ability of the valves to close; the effect of distension of this, the weakest portion of the heart. I suppose the same pulsation would be noticed in the pulmonary veins, if they could be seen or felt, in cases of regurgitation through the orifice of the mitral valve.

The practical inferences to be drawn from this study of the physical signs of valvular disease are these:

You may have valvular disease without any physical sign; without endocardial murmur. This may happen when the disease is trifling. It may happen when the action of the heart is feeble, especially towards the close of the disease. Finally, it may happen when certain forms of valvular disease are present much more readily than in other cases. Obstruction of the ori-

fice of the tricuspid valve, of that of the mitral valve, are the least apt to be attended with an endocardial sound; while obstruction of the aortic orifice, imperfect closure of the same orifice, or of the orifice of the mitral valves, are usually accompanied by a distinct endocardial sound; and the importance of this fact is much increased by remembering that these valves are by far the most frequent seats of disease.

In the second place, you may have an endocardial sound and no valvular disease. But this will happen only in relation to one form of endocardial sound, that belonging to obstruction of the aortic orifice. Attention to the facts I have already stated, will enable you to distinguish the inorganic from the organic sound.

The sum of all is this: in cases in which decided valvular disease exists, you can usually, with care and attention, ascertain not only its existence, but even its precise seat and character. But in order to do this successfully, you must never be in a hurry. You should examine the heart several times, and under the different circumstances of comparative excitement and quiet. Experience has taught me the value of this rule—never to be in haste in the diagnosis of valvular disease. It frequently happens that the rhythm of the heart is so much disturbed, that it is impossible to determine, at a first examination, whether the endocardial sound accompanies or masks the first or the second sound of the heart. A mistake on this point would, of course, defeat all accuracy in the diagnosis. The same caution will enable you also to ascertain whether the endocardial sound is permanent or intermittent; whether it is constantly rough, or occasionally soft and blowing—all points of importance in establishing the true diagnosis of valvular disease.

Occasionally, in cases of valvular disease of the heart, you can feel a thrill communicated to the fingers over the region of the affected valve. This fact adds nothing either to the certainty or to the accuracy of the diagnosis. It only teaches that the vibrations, which are often strong enough to be heard, are sometimes also strong enough to be felt.

I have dwelt with considerable detail upon the endocardial sound as indicative of valvular disease; and I trust that I have

not only placed the subject before you in an intelligible manner, but also in a true light, according to the present state of the science of auscultation. Much misapprehension and doubt has existed on this subject in the minds of many intelligent observers. This has arisen, in part at least, I think, from expecting too much—too much uniformity and certainty in the physical signs. A want, also, of sufficient attention to all the circumstances that influence the endocardial sound, especially the action of the heart at the time of examination, may have contributed to the same result. There can be no doubt that enlargement of the heart is far more certain and obvious in its physical diagnosis than valvular disease: it is far more important also. The enlargement is the chief thing; the valvular disease is but an appendix, or sometimes a preface, to the great work of mischief, and which might be struck out without altering in any very important, certainly in no essential respect, the most marked features of the case.

The attempt has been made to gain some assistance in the diagnosis of valvular disease, especially of the left side of the heart, from the condition of the pulse. I have already alluded to the same attempt in the diagnosis of enlargement of the heart, and I then stated, that although you may sometimes gain assistance from this source, yet that you will be often disappointed. The same thing is true in valvular disease. In cases in which the aortic orifice is much obstructed, you might naturally expect to find the pulse small. In cases in which there is aortic regurgitation, you may find it small and feeble, and perhaps notice a back stroke, especially if the regurgitation is considerable. Imperfect closure of the aortic orifice is also supposed to induce a thrill and increased pulsation in the arteries of the neck, such as are noticed in cases of anemia, and which, in both cases, may be owing to unfilled arteries—an explanation offered by Dr. Corrigan, of Dublin. In a case that recently fell under my observation, in which increased pulsation, thrill, and even a sawing sound was heard in the arteries of the neck, the signs of slight aortic obstruction, and still more marked, of aortic regurgitation were present. Finally, in mitral regurgitation, the pulse is often found feeble and small, as if the artery did not contain its

full supply of blood. While I readily admit these conditions of the pulse in the different forms of valvular disease of the left side of the heart, yet I contend that none of them are constant or characteristic. Indeed, it is easy to see that the condition of the muscular structure of the heart and the degree of valvular difficulty, must modify the state of the pulse.

In connection with valvular disease of the heart, I may mention a congenital disease which sometimes impresses a peculiar aspect upon the case, by inducing a remarkable blueness of the skin. In most cases of disease of the heart, you will notice a slight tendency to lividity, especially of the face; and when, from any accidental cause, the obstruction to the circulation becomes unusually great, as by the supervention of bronchitis or pneumonia, this lividity becomes a striking feature of the case. But in cases in which the communication between the right and the left sides of the heart continues open through the foramen ovale, or in which, from malformation, the two ventricles communicate by an opening, or in which the pulmonary artery and aorta spring from a common origin, so that there is not only an impediment to the circulation, but an actual mixture of the venous and arterial blood in the heart; this blueness sometimes assumes an extraordinary degree of distinctness. It has even been regarded as a disease by itself, and called *morbus cerulæus*. It is attended, however, with enlargement of the heart, like valvular disease, and cannot, properly, be separated from it. Indeed, the observations of Louis seem to prove that the mere communication of the two sides of the heart through the foramen ovale, does not produce the remarkable blueness you notice, until the right auricle has gained so much power over the left, by hypertrophy, as to force a portion of the venous blood through the opening into the left auricle. It must be evident, that while the auricles themselves possess the same power, and act at the same time, which they do, naturally, there will be no mixture of the blood through the foramen ovale. The pressure being equal on each side of the opening, the blood will not pass in either direction, but will flow onward through the natural channels, provided there be no obstruction. But that such obstruction does exist, is proved by your always finding enlargement

of the right side of the heart in fatal cases. Sometimes the cause is apparent, as a narrowing of the mitral orifice; in most cases, however, it escapes observation.

Another reason why you should suppose that the whole difficulty is not congenital is the fact, that the blueness does not always show itself early in life; sometimes, indeed, only after an interval of many years. In this case, the congenital communication between the two sides of the heart is not enough to induce the blueness; but in time, from some obstruction to the circulation, the right side of the heart enlarges, gains new power, and a portion of the venous blood is forced into the left auricle to contaminate the arterial circulation. Indeed, it is not uncommon to find the foramen ovale partially open in adults, who have presented no symptoms of disease of the heart. In these cases, no mixture of the two kinds of blood is supposable.

The symptoms of this form of disease are those of enlargement of the heart. The blueness, which ordinarily is confined to the face, but sometimes extends, more or less, over the body, is the most striking symptom. When the patient is quiet, the blueness often subsides in a great degree, but under any excitement, as from anger, or from exercise, the color immediately heightens, and sometimes to a degree really astonishing. Such patients are observed to be remarkably prone to fainting, and often die suddenly. They are also much less subject to dropsical effusions than others suffering from disease of the heart. The reason of this is not apparent, unless it be that the obstruction to the circulation on the venous side of the heart is partially relieved by the passage of a portion of its blood to the arterial side.

In the physical examination of such cases, you will find the usual signs of enlargement of the heart, and perhaps those of valvular disease. But I am not aware that the passage of the blood through the foramen ovale has ever been noticed to generate an endocardial sound. If this is sometimes the case, and I see no reason why it might not occur, it must resemble in its general character the other endocardial sounds. Its precise diagnosis, however, must be left to actual observation.

LECTURE XXXI.

DIAGNOSIS OF ENLARGEMENT OF THE HEART.

Enlargement of the heart, as distinguished from chronic serous effusion into the pericardium, from aneurism of the thoracic aorta, from pulmonary emphysema. —Functional derangement of the heart, as caused by dyspepsia, venereal excess, the use of tobacco and coffee, and by spinal disease.—Angina pectoris, from functional derangement of the heart.

The different symptoms and physical signs that have been enumerated, embrace all the important facts in the history and diagnosis of enlargement of the heart, whether it exists as a simple disease, or as connected with important changes in the structure of the valves and orifices of the heart. Studied separately, or in the order of their importance, as they affect the functions of the organ itself, or those of remote organs, or the constitution generally, they present you with the materials of diagnosis. Unfortunately, however, the definiteness which you may find in a book or in a lecture is not always met with at the bedside. New combinations are constantly presenting themselves, which perplex the judgment and fill the mind with doubts: thus, clinical medicine never reaches perfection, even after a long life of study and observation; each case of disease is an individual case, having its own peculiarities.

Still, the different phases of a disease which rests upon the solid basis of a sound pathology, if incapable of accurate classification, can yet be thrown into groups, by which its study is simplified and its diagnosis is more clearly established.

Enlargement of the heart, whether preceded by valvular disease or not, is almost always a chronic affection, even in cases in which the attack seems sudden. Although it may happen that the first suspicion of heart disease may arise after a paroxysm of dyspnœa, or an attack of angina pectoris, yet a careful examination will detect that this is not the real beginning of the disease. Such patients have been sensible, often for a long time,

of slight dyspnœa on going up stairs, or of palpitation, or of being easily fatigued.

In cases in which valvular disease is the first step in the series of morbid changes, you might naturally expect that the disease would be more rapid in its progress, more strongly marked in its symptoms, than in cases of simple enlargement. This is true in a majority of instances; yet so varied is the ability to resist disease in different constitutions, so different is the power of adaptation, if I may so speak, to unfavorable circumstances, that cases, the most discouraging in their first aspect, may agreeably disappoint your expectations. Thus, after an attack of severe rheumatic inflammation of the heart, the physical signs of serious valvular disease may continue, and yet the patient apparently recover entirely, or suffer so little from symptoms of cardiac disturbance, as to be able to enjoy life, and to perform its duties. Year after year may pass on, and no increase of difficulty ensue, until, finally, the usual train of cardiac symptoms develop themselves, and lead, more or less rapidly, to a fatal issue. Such cases, while they are witnesses of our progress in the art of diagnosis, are yet most melancholy evidences of the imperfection of our therapeutical resources. How often does it happen among the young and vigorous subjects of acute rheumatism, who have apparently recovered perfect health, that we find, by auscultation, the undoubted traces of valvular disease, which art cannot remove, and time will only aggravate!

But whether organic disease of the heart commences as a rheumatic affection in the young, or from some more obscure cause in those advanced in life, its full development usually presents unequivocal evidences of its existence. Dyspnœa after exercise, palpitation, and perhaps pain about the precordial region, are present. An exploration of the chest discloses the displacement of the apex, increased dulness on percussion, extended impulse, and, perhaps, other more equivocal signs, as evidences of enlargement of heart. While, if valvular disease is present, the blowing sound, in some of its modifications, may point out its precise seat and character. With these local indications, the pulse will be found to be often accelerated, sometimes unnaturally slow and laboring, sometimes small or feeble,

and perhaps irregular. The patient will often complain of flushings, of pain and of throbbing in the head, increased irritability of temper—symptoms of active cerebral congestion. Symptoms of angina pectoris may occasionally occur, and uneasy sensations after meals, from distension of the stomach by food, and especially by flatulence. The countenance may present only the appearance of florid health, or you may notice a lividity about the lips, and an anxious expression about the brow and eye. During the more early period of even confirmed disease, if the patient can limit the circle of his actions, he may pass on with but trifling inconvenience—but a single step beyond that narrowed circle forcibly reminds him of his true situation. As the disease advances, the symptoms already existing increase, especially the dyspnœa and the precordial distress, while the palpitation may diminish. The countenance becomes pale, livid, bloated, and the look of anxiety more marked. Emaciation progresses, while the feet begin to show indications of dropsical effusion, subsiding again, perhaps, for a time, but, finally, extending upward to the abdomen, or to the chest. In this stage of the disease you can also frequently feel the liver enlarged by congestion, extending below the false ribs. Bronchitis, indicated by cough and by the sibilant rhonchus, and flatulence, aggravate the distress. The pulse becomes more feeble, the extremities inclined to coldness, until, at length, orthopnœa and inability to sleep indicate the near approach of that agony which, finally, renders death welcome. The physical examination of the chest, in the advanced stage, is often more difficult than at an earlier period. As the vital powers fail and the action of the heart diminishes in force, it is more difficult to detect the point where the apex strikes, and the signs of valvular disease, from the same cause, and often, also, from irregularity in the rhythm of the heart, lose their distinctness, or may cease altogether.

The mode of death in enlargement of the heart is by no means uniform. The most common, and, if I may use the expression, the most natural mode of termination, is that I have already mentioned, when the patient dies gradually exhausted, after a prolonged and most distressing agony. But he may die sud-

denly, in a paroxysm of angina pectoris, or from rupture of the heart, or from a polypus obstructing one of the orifices, or he may become comatose, or pass into a typhoid state. Sudden death in disease of the heart is a popular belief, and often excites a good deal of anxiety in patients. But it should be known that but few, comparatively, die in this way, and in these cases the sudden termination is often the fault of the sufferer. A paroxysm of passion, a violent and sudden physical effort, improper indulgence at the table, will often be found to be the direct cause of danger, and few die at last from these causes without having had, in their own experience, repeated warnings of the risk they incur.

Enlargement of the heart is sometimes a latent affection, at least during the earlier periods of its progress. I recollect, many years ago, attending a young man with pneumonia, and finding, accidentally, when examining the chest, undoubted evidences of enlargement of the heart. At the time, I made the most careful inquiries as to the previous evidences of a disturbed state of the circulation, but could find none whatever. After a time, however, the same patient called upon me with all the symptoms of enlargement of the heart fully developed. I have since met with similar cases, in which the heart was enlarged without symptoms. They are usually, I think, cases of simple enlargement. Indeed, it is easy to conceive, when the obstruction of the circulation is at some remote point, and moderate in degree, that a moderate enlargement of the heart, by giving it a little more force, may prevent the symptoms of obstructed circulation, rather than operate to cause them. Just as in the gradual enlargement of the heart occurring, universally, in advanced life, and when the arterial system has lost something of its natural elasticity, the organ, a true balance-wheel, tends to prevent the symptoms of a retarded circulation. I have alluded to this subject in a former lecture.

A gentleman, about thirty years old, and in the previous enjoyment of good health, had an attack of remittent fever three months before I saw him. During his convalescence he relapsed, and failed gradually. A slight cough ensued, with a tendency to diarrhœa. The symptoms resembled those of phthisis, but no

physical signs of disease of the lungs could be detected. He complained of pain in the right lumbar region, and of other symptoms which led to the suspicion that disease of the kidney existed. During a careful examination of his symptoms, nothing occurred to give me the slightest suspicion that any disease of the heart existed. Several other physicians saw the patient, and none of them suspected the existence of any such disease. After the death of the patient, which happened soon, the heart was found much enlarged—the lungs were healthy. An abscess existed in the right kidney. I have no doubt, in this case, that had the attention been drawn to the heart, the physical signs of enlargement would have been noticed. The case is not without its instructive point, teaching you the importance, in all cases, of examining the physical condition of the different organs as thoroughly as possible, even when no symptoms of disease are present.

There is another cause which renders disease latent, and here the fault is in the physician and not in the patient; and this is, when the mind becomes preoccupied with the idea that a certain disease exists, either from the statement of another physician in whom you have confidence, or from other reasons.

I recollect being called to see a lady in consultation, who had lost a sister with phthisis, and had been herself disposed to pulmonary disease. She was about five months advanced in pregnancy. A month before her pregnancy, she began to have a dry cough, with dyspnœa, and with a disposition to chills. She had several times expectorated a moderate quantity of pure blood. For two or three weeks she had suffered from profuse night-sweats, and for a fortnight her legs had swelled a good deal. The pulse was 106. She had for a long time suffered from palpitation, but this symptom was not marked. A sound, resembling the cracked-pot sound, was noticed below the clavicle. The case was considered as a tuberculous affection of the lungs. I saw her only once.

In the progress of the case, the anasarca became very great, and the dyspnœa very distressing; and four days before her death she had an abortion.

In making the post-mortem examination, not a tubercle was found in the lungs, but these organs were much congested, and contained spots of pulmonary apoplexy. There was abundant hydrothorax on the right side. The heart was the principal, and no doubt the original, seat of disease. It was generally enlarged, more dilated than hypertrophied, and the right side more affected than the left side. The pulmonary orifice was large and free, and the pulmonary valves hypertrophied, as were also the tricuspid valves. The aortic orifice and valves were healthy, but the mitral valve was bone-like, and the orifice reduced to a mere chink. An old polypus existed in the cavity of the left auricle. A considerable quantity of serum was found in the left pleura.

It is not probable in this case, that any morbid sound existed over the heart, for the blowing sound is sometimes absent in this form of valvular disease, as I have already stated; but an examination of the precordial region would, I think, have detected an enlarged heart, and this was not done. There is one point that should be noticed in this case. The patient was constantly growing worse as her pregnancy advanced, a condition more allied to diseased heart than to tuberculous lungs.

Chronic pericarditis, especially in that form called essentially chronic, may easily be mistaken for enlargement of the heart. In both diseases, the constitutional and the rational symptoms are so similar, as to afford you no aid in the diagnosis. In both, there is the same gradual development and progress of the symptoms. In both, palpitation, pain, and oppression in the precordia are usually noticed. In both, you have dyspnœa, gradual exhaustion of the vital powers, perhaps irregular pulse, and a tendency to œdema of the lower extremities. In pericarditis, however, you may find an evening exacerbation of fever, and perhaps a well-formed hectic paroxysm, which would not occur in simple enlargement of the heart. When, finally, you come to study the physical signs of the two diseases, you will still find points of resemblance. In both, you will have a circumscribed, yet well-marked dulness over the precordia; in both, you may have dilatation of the same region, and although, in most cases of enlargement of the heart, a displacement of the apex will

guide you to the true diagnosis, yet in cases of very great enlargement, and in which the action of the heart is feeble, it is often difficult to find the apex. These are the cases also in which the sounds of the heart, rendered obscure by the great thickness of its muscular structure, resemble very much what you will notice in effusion into the pericardium.

Dr. Hope has stated that in effusion of serum into the pericardium, the impulse, as noticed over the precordia, is undulatory. Admitting this to be sometimes the fact, for it certainly is not constant, it possesses no diagnostic value, for the same undulatory impulse is sometimes noticed in cases of adhesion of the pericardium, and even in cases of enlargement of the heart. The explanation, however, of this undulatory motion is not the same in these different cases. In effusion into the pericardium, it is caused by the liquid effusion; in the two other forms of disease, probably by a nearer approximation of the heart to the walls of the chest.

Indeed, I think you must admit that a case of chronic pericarditis, or of hydrops pericardii, with moderate effusion of liquid, can only be distinguished from certain cases of advanced and considerable enlargement of the heart, by the greatest attention, and even then, perhaps, not always with certainty. If, however, the effusion be considerable, so as to extend upward to near the clavicle, then the evidence of its existence is much more clear; for I do not believe that the dulness from enlargement of the heart extends above the second rib. Again, when the impulse is so feeble that the hand does not detect it, do not infer from this that effusion exists. Careful examination with the stethoscope may still detect the apex by the shock, and far to the left of its natural position. You must remember always that a chronic liquid effusion into the pericardial sac is a very rare form of disease.

Endocarditis in a chronic form, or rather the valvular lesions consequent upon endocarditis, may induce symptoms resembling those of enlargement of the heart. The symptoms, like those which often occur in simple enlargement of the heart, are symptoms of obstruction to the circulation, and are accompanied by the physical signs of valvular disease, that is, by the blowing sound. Cases may occur, especially after acute rheu-

matism, in which valvular disease is clearly existing, and in which none of the physical signs of enlargement of the heart have as yet appeared.

An aneurism of the aorta may imitate an enlargement of the heart in many of its rational symptoms, although, as you will readily notice when I come to speak of the former disease, there are usually important differences in this respect. The most certain mode to obtain a correct diagnosis, however, is to study the physical signs. In aneurism, the precordial region frequently gives no signs of disease. There are no signs of enlargement of the heart, no evidence of valvular difficulty. There may be increased impulse, but it will have its seat, not over the heart, but in some other portion of the chest. There may be a blowing sound, but it, equally, will not have its maximum over the heart, but at some point more or less remote from it.

Finally, enlargement of the heart is sometimes confounded with emphysema of the lungs, or with what is called asthma. Many cases are called asthma, which are in fact cases of enlargement of the heart. The points of resemblance between these diseases are, the dyspnœa and oppression, with a tendency to bronchitis with extensive and persistent sibilant rhonchus aggravating the dyspnœa into paroxysms. It is true, also, that emphysema naturally leads to enlargement of the heart, and that the two frequently coexist, with an aggravation of the symptoms. In these cases, if dropsical symptoms ensue, you may believe that enlargement of the heart has supervened, for simple emphysema does not seem capable of producing dropsy. The sure method, however, in all cases is to examine the chest, and ascertain whether the physical signs of emphysema, or of enlargement of the heart, or of both conjoined, are present. There is no difficulty in the diagnosis of even these complicated cases with proper attention.

It was remarked by Dr. Hope, that one-half the patients who presented themselves to him with the idea that they were suffering from disease of the heart, were in fact suffering from mere *functional disturbance of that organ*. My own experience fully confirms this opinion. It is not uncommon to meet with cases in which the symptoms present themselves with great

severity, and continue for a long time without organic disease. In such cases, there is something in the history which should lead you to suspect their true nature. The gradual development of the symptoms, and especially their permanence and constancy under the same circumstances, which mark organic disease, are often wanting. It is the character of organic disease of the heart to progress, or at least not to retrograde, under the same circumstances. Active excercise especially aggravates it. While in functional disturbance, the symptoms may disappear even for a time, and return at irregular intervals. It is a striking fact, that active exercise, which so uniformly acts unfavorably on the symptoms of organic disease of the heart, often acts in precisely the opposite way in functional disturbance of that organ. Thus, it is not uncommon for the latter class of patients to express themselves as feeling better after a long walk, or after a journey, in which the mind and body have been actively engaged. The dyspnœa, which forms so constant a symptom in the organic cases, may be entirely wanting, at least at intervals, in the cases of functional disturbance, or present itself in so slight a degree as to occasion no inconvenience. Patients thus affected will sometimes run up stairs, even without difficult breathing, while palpitation and precordial distress are very prominent symptoms. As a general rule, dropsical symptoms do not occur in these cases. I have noticed them occasionally in cases of chlorosis, in the feet and legs, but I think never in the serous cavities. While those symptoms, which are called nervous, as pain in the head, neuralgia in different parts of the body, depression of spirits, coldness of the extremities, are often so prominent as to add greatly to the sufferings of the patient. Indeed, I am inclined to think that there is generally more distress in the cases of functional disturbance of the heart, than in those of an organic nature, at least during the earlier progress of the latter cases.

But whatever doubts may have arisen in your minds during the study of the symptoms of such cases, they should vanish at once when you come to examine the precordial region. I have already remarked that, in organic disease of the heart, symptoms do not usually attract much attention before enlargement

of the organ exists. At all events, if the symptoms have continued any length of time, as for instance for several months, you should certainly expect to find the signs of enlargement. Now, in the cases of functional disturbance, even if they have existed for years, no physical signs of enlargement exist. The apex strikes in its natural position; the impulse may be increased in degree, but not in extent; percussion over the heart is natural. You may, indeed, hear a blowing sound over the aortic valves in certain cases, especially in chlorosis; but this, if sufficient care be used, can hardly deceive you. It always accompanies the first sound of the heart; it extends to the arteries and veins* of the neck; it is intermittent. These signs, taken in connection with the general history of the case, and especially with the absence of all the signs of enlargement of the heart, will easily guide you to a correct diagnosis. In functional disturbance, the sounds of the heart are usually distinct, sometimes to a greater degree than is natural; and so they may be in organic disease of that organ. The greater or less clearness of these sounds does not appear to me to have much diagnostic value.

It is interesting to trace the causes of this functional disturbance of the heart. In many cases you can refer it to dyspepsia, although when this is the true cause it is not always easy of detection. It is a well-known fact, that when the irritation of one organ, as the stomach, acts secondarily upon another organ, as the heart, the symptoms of the primary affection are more or less masked. Thus, although the stomach may be the original seat of the attack, yet great care is often requisite in detecting its true condition. Patients are sometimes ignorant of the existence of dyspeptic symptoms, while a careful study of the way in which the functions of the stomach are performed, and especially of the effect of remedies, will clearly teach you that gastric derangement exists. In cases of chlorosis, the cause of the symptoms is more readily recognized.

Cases of functional disturbance of the heart are occasionally dependent on irritation of the spinal column in its thoracic por-

* In chlorosis and in anemia, a continuous sound, called by Dr. Hope the venous hum, from its supposed seat in the jugular veins, is sometimes noticed.

tion. I have known it follow a fall, in which this portion of the spine was affected.

Venereal excesses are a frequent source of disturbance of the heart. Seminal emissions, from the habit of masturbation, are often at the bottom of the difficulty, and much tact is necessary to elicit the truth. The patient seldom makes a voluntary confession; indeed, he is sometimes ignorant of the true cause of his symptoms. Such cases wear a peculiar aspect, which will awaken the suspicion of the experienced observer as to their true character. The same symptoms are sometimes noticed in married persons, where a husband of rather feeble powers is united to a vigorous and exacting wife. Indeed, any cause tending to exhaust the nervous energies may lead to the same result. The inordinate use of tobacco, or of strong coffee, long-continued mental anxiety, undue mental effort, if united with sedentary habits, will produce the same effect. I have frequently noticed the same symptoms in students, especially towards the close of the session, and which exercise and country air would soon remove. Finally, there are cases in which you may feel sure that the disturbance of the heart is merely functional, but in which the cause escapes you. The removal of the apprehension of organic disease, however, is a great relief, if not an important step towards the cure of such affections.

Cases of chlorosis, when complicated with heart symptoms, are those which are distinguished from disease of the heart with the greatest difficulty. I recollect some time ago visiting a young lady with a physician of this city, who was supposed to be laboring under a disease of the heart, and indeed the symptoms were very much like it. The patient was almost confined to the bed; she was pale and bloated, and complaining a great deal of palpitation and dyspnœa, and with the lower extremities very œdematous. She had suffered from these symptoms, I think, for some months, and she appeared like a person far advanced in organic disease of the heart. Yet a careful examination of the precordial region could detect no symptoms of enlargement of the heart. Relying on this fact alone, I ventured to express the opinion that the case was one of chlorosis, and recommended the free use of iron. The patient soon recovered.

The cases which occur in connection with improper venereal indulgences are very common in my experience. Many striking cases have occurred to me. I recollect the case of a young gentleman of rather delicate constitution, about twenty years of age, who had been addicted to masturbation from the age of puberty, and was, at the time I first saw him, troubled with seminal emissions at night, as often as once a week. His physician had told him that he was suffering from disease of the heart, and that he might die suddenly with it. He had also treated him by blood-letting, and low diet, and had urged as much quiet and rest as possible. The poor patient, with his symptoms aggravated by the treatment, and his mind distressed by the apprehension of a disease which he thought might terminate his career at any moment, was in a most pitiable situation. His whole nervous system was highly excited. He complained of palpitation and of some dyspnœa. I carefully examined the precordial region, and although his symptoms had long been present, a year or longer, I could not find the least evidence of an enlarged heart. There was accelerated action—nothing more. I did not hesitate to assure the patient that he had no disease of the heart, but it was difficult to persuade him, in his state of nervous excitement, that this was the truth. By persuading him to take exercise, at first moderately in a carriage, and afterwards on horseback, by regulating his diet, by the use of iron and other tonics, his heart symptoms gradually left him, and for a long time I have heard nothing about them. The tendency to nocturnal emissions, however, continues, notwithstanding the employment of various means to arrest them, and among other means, cauterization of the urethra.

I remember another case occurring in a young Portuguese, 19 years of age, who had been for several years in the habit of masturbation, once a day. He had, however, abandoned the habit three months since, and was not troubled with seminal emissions. About six weeks before I saw him, while eating his breakfast, he was suddenly seized with a paroxysm of dyspnœa and of palpitation, which continued about a quarter of an hour. Since that time he has had five or six similar paroxysms. In the intermediate periods he has felt rather weak, his appetite

has been impaired, his digestion imperfect, and his bowels deranged. He thinks that his breathing is a little oppressed in going up stairs. One evening, after having felt as well as usual during the day, and while he was sitting quietly reading after tea, he was suddenly seized with a sensation like a flush of heat in the head and in the chest, followed by violent palpitation and by dyspnœa, which did not continue longer than a quarter of a minute, but gave way to a rapid, fluttering, irregular action of the heart, accompanied by coldness of the extremities, and by moderate dyspnœa. There was no pain about the heart. When I saw him, two hours after the attack, he was tranquil, his respiration easy, the action of the heart accelerated, but regular, and with the impulse moderate in degree. A careful examination of the precordial region discovered no signs of enlargement of the heart, no abnormal sounds.

The suddenness of the paroxysms in this case, and their appearance when the patient was most quiet, were certainly indicative of functional disturbance, rather than of organic disease of the heart. The suddenness of the paroxysm is not, however, so diagnostic as the attack during a period of rest. For I have seen, within the last year, two cases of organic disease of the heart, both of which have terminated fatally, and in which a post-mortem examination was made to confirm the diagnosis, in which a sudden paroxysm of dyspnœa was the first symptom of disease of the heart that attracted attention. One of the cases was that of a well-known comedian, who was suddenly attacked while on the stage. In the other instance, I think the gentleman had risen at night to open a window.

In the case that I will next detail, symptoms resembling angina pectoris were present. The only cause for the symptoms that could be detected, was a buxom wife, aided, perhaps, by too great application to his trade. At all events, a treatment founded on these suppositions was perfectly successful.

A baker, 30 years of age, a pale-looking man, enjoyed good health until about seven years ago, when he began to be affected by palpitation and by dyspnœa. After a time he began to experience a gnawing sensation about the lower portion of the sternum, extending across the base of the chest. Finally, the pain extended

up to the neck and jaw, to the top of the shoulders, down the arms, and also down the legs, in succession. He compared this pain throughout to a "gnawing soreness." It never attacked him while in bed, but invariably when using active exercise. During the time it continued, which may be fifteen minutes, there was also a sense of suffocation, and the action of the heart became very feeble, or sometimes it seemed to cease beating. As the paroxysm passed away, the action of the heart was increased. He had also paroxysms of determination of blood to the head, caused also by excitement, and marked by flushing of the face, distension of the temporal veins, pain and dizziness in the head, but there was no appearance of lividity. The action of the heart was feeble, and very much accelerated: I counted 208 pulsations in a minute, while it was evident that not half that number of pulsations reached the wrist; for the pulse there was only 96 in a minute. There was also, occasionally, an irregularity, an intermission in the rhythm of the heart. There was no physical sign of enlargement of the heart, no sign of valvular disease. The patient was evidently feeble, and his countenance was pale, but he had continued his occupation until recently. There were no dropsical symptoms. The digestive functions have been well performed until the last two weeks, with the exception of some flatulence, and a tendency to constipation.

This patient entirely recovered by being separated from his wife, and by going into the country and using the preparations of iron.

The symptoms in this case bore a considerable resemblance to those of angina pectoris, and, like them, were excited by active exercise, especially by going up stairs. They had existed for seven years, and yet no decided enlargement of the heart was manifest—a fact, in itself, enough to prove the absence of organic disease of the heart.

A physician, aged about thirty-five years, practising in the country, of robust constitution, was struck down by lightning eleven years ago. During some months after this accident he suffered much from pain in the head, and experienced an attack of cerebral congestion so severe as to resemble apoplexy, from which, however, he perfectly recovered. Before the accident

by lightning, he had noticed a remarkable ability to withstand the action of electricity from the machine, but since that time the least shock overpowers him. He continued to enjoy good health until about four months ago, except that for several years he had occasionally felt a soreness at the lower part of the sternum. Four months ago he accidentally slipped upon the ice, and made use of uncommon efforts to recover himself. The same night he was seized with severe symptoms resembling those of angina pectoris. Ever since that time he has experienced pain and soreness along the spine between the shoulders. The paroxysms of angina have continued to recur, and attack him after unusual exertion or fatigue. They sometimes also occur when he is quiet in bed, and with equal severity.

He is seized with excruciating pain in the precordia, with constriction. The pain shoots down the left arm, along the track of the ulnar nerve, and occasionally, with less severity, down the right arm. It is accompanied by numbness, by a sensation of tingling, and by a temporary paralysis of the left arm; but there is no lividity. He has noticed no sense of suffocation except in two or three paroxysms, and compares the sensation to that produced by the inhalation of nitric acid. During the paroxysm, the pulse in the left radial artery becomes extinct, and very feeble in the right radial artery. The paroxysms pass away with a copious discharge of urine. He also complains of more or less shooting pain along the intercostal nerves of the left side, and lately the nerves passing up the back of the neck to the head have been similarly affected.

The appearance of this gentleman was that of perfect health, with this exception, perhaps, that the countenance was rather pale. His digestion is good, his nervous system is not affected, except locally, as just mentioned. The pulse was soft and feeble, and stronger in the right than in the left wrist (this may be congenital).* The sounds of the heart are natural. There are no signs of enlargement, or of valvular disease. The action is feeble, and occasionally intermittent.

* It is possible, in this case, that there may be a latent aneurism of the thoracic aorta.

The two superior dorsal vertebræ are prominent, and evidently enlarged, and there is tenderness on pressure, most marked over the transverse processes. The same tenderness exists over the vertebræ below, and between the blades of the scapulæ, where there is no enlargement. The tenderness on pressure is more marked over the transverse processes of the left side than over those of the right side. The same tenderness exists over the left intercostal nerves in the axillary region, and along the left border of the sternum. This gentleman had been bled largely, and had taken the most powerful narcotics, but without the least benefit.

I looked upon this case as one of neuralgia of the heart and of the spinal nerves, dependent upon disease of the vertebræ, or, at least, of the ligaments and tissues immediately connected with the vertebræ. I recommended an issue to the spine, and other remedies in accordance with this view of the case. I regret that I cannot report the subsequent history of the case.

Cases like that just mentioned undoubtedly occur, in which the real disease is in the spinal column. I recollect a case, as detailed to me by one of the friends of the patient, to which two distinguished physicians attended for a long time, and regarding the case as one of organic disease of the heart, treated it accordingly. The patient grew worse, and, finally, all hope of improvement was abandoned. At length, however, a different physician was called in, who, fortunately, took a different view of the case, and introduced an issue over the dorsal vertebræ, and at the same time used tonic remedies. The rapid recovery of the patient, and the subsequent good health, proved, clearly, that the affection of the heart was entirely sympathetic.

LECTURE XXXII.

ORGANIC DISEASE OF THE HEART.

Treatment of organic disease of the heart.—Treatment of enlargement of the heart and of valvular disease.—Blood-letting, purgatives, diuretics.—Diet and regimen.—Treatment of functional derangement of the heart.

The treatment of enlargement of the heart may be reduced to the fulfilment of a single indication—that of equalizing the circulation of the blood. In health, the heart may be regarded as a regulator, by which each organ is made to receive its due supply of blood, and afterwards to return it to the general circulation. The moment any portion of the heart becomes unequal to the performance of its duties, whether this arise from increased or from diminished power, a tendency to congestion in some part of the system takes place. In many cases it is easy to ascertain that both these causes of congestion exist at the same time, that while the one portion of heart is forcing the blood too rapidly into the system, another portion is obstructing its progress. In most cases, it is probable that obstruction is the primary evil, residing either in the heart itself, or in some remote organ, and that increased action is the result. But it is not always easy to establish the fact. Those organs which are more immediately under the influence of the action of the heart are the earliest to feel the effects of this congestion. Of these organs, the lungs hold the first place, then the brain, then the liver.

There are two modes of equalizing the circulation in cases of enlargement of the heart. The one, direct, by diminishing the amount of the circulating mass by blood-letting in some form; the other, indirect, by limiting the exercise of the patient, both mental and physical, by a simple diet, and lastly, by relieving the tendency to congestion in certain organs, by exciting the functions of other organs in which no such tendency exists, especially by purgatives and by diuretics.

The first method, that by blood-letting, is the most direct and simple. By diminishing the quantity of blood, you lessen the effects both of obstruction and of increased action. The heart, like an overladen animal relieved of its burden, moves on again more steadily and quietly. Indeed, the relief often experienced in these cases, is the most complete that you can be called upon to witness in the whole course of your clinical experience. And yet it is only in certain cases that such a decided remedy as blood-letting is useful or proper. In the earlier period of heart disease, when symptoms of active congestion of the brain or of the lungs exist, when the patient is plethoric and the vital powers not much impaired, then it is that a free venesection will produce almost immediate relief. In other cases, in which the patient is more feeble, or the symptoms are not so urgent, the application of cups to the precordial region, or to the temples, or to the chest, will be equally beneficial. The danger is, that the benefits of bleeding in the earlier stage of the disease, may tempt you to carry it too far, or to repeat it too often. If you pursue this method, you will soon find that the remedy loses its power, or becomes injurious by the debility and reaction it induces. There are many patients who are much benefited by the occasional loss of a small quantity of blood, a few ounces from the precordial region, not only during the earlier stage of the disease, but even at a later period. This small quantity will often produce great relief, to your surprise, if you have been accustomed to resort to free venesection. When increased palpitation, dyspnœa, pain in the head, or other symptoms indicate a tendency to internal congestion, this small local bleeding will often answer every purpose of relief, and without any risk of unpleasant consequences, from debility, or from reaction.

The indications for blood-letting, then, are—the strength of the constitution, the stage of the disease, the fulness of the blood-vessels, and, I may add, the violence of the symptoms of congestion which attend these conditions. In a few cases, the rapid and free abstraction of blood by venesection is indicated; but in many more, the local abstraction of a few ounces of blood is the true indication.

In the advanced stage of disease of the heart, blood-lettting

from the arm is generally inadmissible, and even local depletion fails to relieve. You must carefully discriminate between cases of the same disease in different stages of their progress. A worn-out heart and an enfeebled constitution, do not bear bleeding; they require rather a tonic or supporting treatment. In such cases, however, you can sometimes effectually relieve the embarrassed circulation and the effects of congestion, by calling into activity the functions of remote organs which are not so liable to congestion. Thus, by acting freely on the intestinal canal and on the kidneys, you can often relieve the heart and other suffering organs.

The effects of purgatives, especially of those of an active, drastic character, in relieving the symptoms of congestion, are often most happy to witness. They are useful in the early stage of the disease, as derivative and depletory in their action, and become useful adjuvants to blood-letting in many cases; but it is at a later period, especially when dropsical symptoms have ensued, that their effects are most happy. The full and free operation of purgatives is often attended with a striking sensation of general relief, and its repetition is soon followed by a subsidence of the dropsical effusion, by improved respiration, happier spirits, and other indications of a favorable change. Purgatives are less debilitating in their effects than blood-letting, although they require much care in their use in weakened constitutions. They hold a middle place, in this respect, between blood-letting and diuretics.

The purgatives most commonly employed in this Hospital are, the compound jalap powder, and the super-tartrate of potassa, the Croton oil, and a pill of scammony and gamboge. The object is to induce free and watery evacuations with as little irritation to the stomach and intestinal canal as possible. The purgative may be repeated every day, or every second day, or less frequently, according to circumstances.

Elaterium is a remedy much recommended as a purgative in cases of enlargement of the heart, but I have not often found it available, on account of the nausea and vomiting it is so apt to induce; so that, after repeated trials, I have been obliged to give up its use. When, however, it does not disturb the stom-

ach, it will act most powerfully and effectively in removing internal congestions. Indeed, so valuable have I sometimes found it, that I have used the utmost care to guard against its effects on the stomach, by administering it in very minute doses, even in doses of the one twenty-fourth part of a grain, frequently repeated, but still it has disturbed the stomach. I have been informed, that if combined with a small quantity of opium, and if rubbed up well with powdered gum-arabic, its unpleasant effects on the stomach may be avoided. In a single case this effect was not produced.

Another class of remedies much employed to relieve internal congestion and dropsical effusions in cases of an enlargement of the heart, are diuretics. These agents are more slow in their operation than purgatives, but less weakening in their effects. They form the chief medical means of relief in the more advanced stage of enlargement of the heart, and at an earlier period when much prostration exists. As a general rule, it is well to precede their administration by the exhibition of a purgative, or even by local blood-letting, if the case requires it. While I wish you to guard against weakening an already exhausted patient by too free depletion, I wish you to be equally guarded against that false weakness which is the result of oppression. The patient, if I may so speak, being kept down by the weight that is upon him rather than by any real debility, take off that weight, which is congestion, by abstracting blood, by purgatives, and your patient rises up at once, refreshed and invigorated. But do not mistake for this temporary depression the real weakness which is the result of the general progress of the disease, the gradual exhaustion both of the heart and of the vital powers. Depletion will not relieve this, even temporarily, but will add to it. The application of the more active means of treatment to cases of enlargement of the heart, is a matter of much tact and discrimination.

The diuretic most frequently employed in this Hospital is, the compound squill pill—calomel, squills, digitalis, each one grain—to be used twice or thrice a day. This is an old and much used formula for dropsical effusions connected with disease of the heart. I have, however, found that it frequently

salivated patients very early, and before its diuretic effects were established. Experience has taught me, in such cases, to look upon salivation as a most unfortunate occurrence. It compels me to stop at once the use of the remedy. If it goes beyond a very moderate degree, its effects are most injurious. It increases the irritability of the system, it excites fever, it induces debility, and, in every way, aggravates the unhappy condition of the patient. Indeed, I am satisfied that I have seen more than one patient with enlargement of the heart, hurried out of existence by being salivated profusely. This experience has induced me to substitute a grain of blue mass for the grain of calomel, and even with this change, I feel obliged to watch patients very carefully. It has appeared to me that dropsical patients are very easily salivated.

It seldom happens that diuretic remedies are used singly. Single remedies, however, sometimes exert great power; and this, I think, is especially true with digitalis. It has been much employed in the early stage of the disease, to quiet inordinate action of the heart. That it does produce this effect is very certain, but it cannot be long continued, owing to its poisonous properties. As a diuretic, however, when the action of the heart is not too feeble, its good effects are conspicuous, especially when given in the form of the infusion.

There are several old diuretic formulæ which have long been employed in this Hospital with advantage. The Diuretic Decoction, composed of squills, seneka, juniper berries, and a little sweet spirits of nitre, is that most frequently employed. Another formula, much older, I believe, and very old-womanish, but which is still, sometimes, very efficacious, consists of horse-radish root, white mustard-seed, and rust of iron, boiled in cider. I have, I think, improved this formula by substituting the acetate of iron for the rust, and adding the acetate of potassa. Another formula, of country origin, but which an experienced practitioner informed me he had found more useful than any other, is composed of milk-weed root, horse-radish root, dandelion root, mustard-seed, juniper berries, and elder bark, boiled in cider. Other remedies have been found diuretic in these cases. Parsley root, the seeds of the pumpkin and of the watermelon,

broom-tops possess this reputation. The super-tartrate of potassa is also much employed, especially, as it forms an agreeable beverage. Fruits, especially melons and peaches; vegetables, especially asparagus, possess diuretic properties which may be useful. The Indian hemp is also sometimes employed with advantage. The iodide of potassium, given with the decoction of sarsaparilla, is sometimes recommended.

The operation of diuretic remedies is proverbially uncertain; one remedy, or a combination of remedies, acting well in particular cases, while they appear to possess no power in apparently similar cases. The cause of this may often escape you, but you must take care that it is not your own fault. If the system is not prepared for their action, they will not afford relief. It may be that the system, especially the circulating system, requires relief by previous blood-letting—it may be that it first requires that its tone should be increased. In either case, diuretics will not act. In the former instance, you have a ready co-agent in the lancet, in local depletion, or in purgatives. In the latter case, unfortunately, your means are neither so prompt nor so efficacious. Exhausted nature will no longer respond readily to the efforts of art.

You are taking a practical view of the symptoms of enlargement of the heart, when you regard them as tending to one result, inability to exercise. This remark embraces both mental and physical exertion, and should never be disregarded, for a moment, in the management of heart disease. Many patients urge on their own destruction by an inattention to this fact. Many cases even of sudden death are owing to active or violent effort; while if the patient is contented to move in the narrow circle his disease allows him, he may hardly be sensible of disease at all. Persons suffering from the early stage of disease of the heart may often enjoy life very highly, with proper prudence, but if they go one step too far, they are suddenly and sometimes fatally arrested in their progress. The limit of exercise will, of course, vary in different cases; but it is safe to say, that whatever excites the heart, or increases the dyspnœa, is beyond the proper mark. Men engaged in active business are frequently attacked with enlargement of the heart, and are

reluctant to quit their customary pursuits. Yet they must be urged to do so, and fortunate are they, if they are in sufficiently easy circumstances to render the sacrifice possible. An active business man, who will retire to the country, perhaps occupying his mind pleasantly with agricultural experiments and with books, or in riding about leisurely in a carriage, will often not only succeed in checking the progress of his disease, but so mitigate his symptoms as hardly to notice their existence; while the poor or less prudent patient, constantly exposed to various causes of excitement, is rapidly hurried onward to a fatal end, perhaps to a sudden catastrophe. Cheerful and moderate exercise, however, is essential to the healthy performance of the functions in this as well as in other chronic diseases.

The same is true also of diet. A too generous diet should be carefully avoided. It fills the blood-vessels, produces distension of the stomach, and thus aggravates the symptoms. Patients with enlargement of the heart almost invariably complain of feeling worse after a full meal. In cases in which the heart is active with a tendency to congestion, a very spare and rigid diet should be enforced. This alone will often supersede the necessity of blood-letting, frequent purgatives, and other more active modes of reducing the system, and is a far better method of controlling the disease. A simple farinaceous diet is the best in some cases, while a moderate indulgence in fish, and the less nutritious kinds of animal food may be indicated as the vital powers begin to fail. In advanced cases, the use of stimulants even may be beneficial. But in all cases in which a more generous diet is called for, you should proceed with great caution, limiting especially the quantity to that which the stomach can easily digest without inducing symptoms of distension. A very common, I think almost constant, symptom in these cases is flatulence; and nothing will guard against its becoming a source of great inconvenience, except a carefully regulated diet. Hence many articles of vegetable diet, which otherwise would fulfil very well the indication of controlling the action of the heart, must be carefully avoided. The same remarks apply also to the condition of the intestinal canal. The accumulation of gas, of indigestible food, an overloaded state of the bowels,

should be carefully avoided, by the administration of mild purgatives, or by the use of injections when necessary.

You will be frequently called to encounter sudden attacks, often of a nature to threaten life, or to mitigate particular symptoms less urgent in their character. You may be called to a paroxysm of angina pectoris, although, more frequently, the violence of the attack will have passed away before your arrival. Yet if you should reach the patient during a more prolonged paroxysm, you should administer, at once, a diffusible stimulant. This will sometimes produce immediate relief, by causing the escape of gas from the stomach, not unfrequently the principal cause of the paroxysm, and by stimulating the heart so as to relieve the congestion of its cavities. Blood-letting, which in such cases becomes an indirect stimulant to the heart by diminishing the load it is called upon to struggle with, is sometimes proper, especially if the patient is plethoric. External stimulants, such as mustard and friction, may also be properly employed, and when the reaction which follows is established, this also should be carefully watched and controlled, if necessary, by the lancet, or by cups, or by the employment of sedatives. These are the cases, above all others, which require a careful attention to diet and regimen during the intervals of the paroxysms. In many cases, it is certainly true that the ordinary symptoms of disease of the heart are not unusually severe, while angina pectoris is of frequent occurrence. This circumstance, however, should not render you less careful in enjoining the greatest prudence, for these cases are really the most dangerous when neglected.

When sudden and violent symptoms of cerebral, or what is more common, of pulmonary congestion ensue, the most prompt means of relief must be employed. Where cerebral apoplexy is threatened, you should at once practise venesection, and that largely, if the patient be plethoric and the vital powers not much exhausted. While I wish to caution you against free depletion, as a general rule, in advanced disease of the heart, yet I know of few cases in which you can, with propriety, carry this remedy so far, as in some cases in which apoplectic symptoms are present. It by no means follows, because the patient has suddenly fallen

insensible, with stertorous breathing, and even with symptoms of paralysis, that actual rupture of a vessel and hemorrhage have ensued. There may be still only congestion present, which a prompt bleeding will entirely relieve. The proof that no hemorrhage has taken place, is in the rapid recovery, especially from the paralysis, which result does not usually occur, and never perhaps perfectly, when the fibres of the brain have been ruptured. Even when hemorrhage has occurred, blood-letting may still be the best means of preventing its increase.

While recommending blood-letting in these cases, I must still caution you against its indiscriminate use. It sometimes happens in apoplectic attacks, that the shock to the nervous system is so great as to paralyze the circulating system. The pulse becomes feeble, the extremities cold. Here early bleeding is not admissible. You must wait for reaction, and favor its appearance by external and internal stimulants, and bleed perhaps afterwards, as the pulse rises and the skin becomes warm. But there are cases, especially the more advanced cases, in which general bleeding is never admissible. Leeches, or cups to the temples or to the neck, may then be indicated, or free purgation, especially by an active injection; with frictions and counter-irritation to the extremities.

Sudden pulmonary congestion is not often so urgent a condition as a similar affection of the brain; because, although pulmonary apoplexy may occur, it is by no means so serious in its consequences as cerebral apoplexy. Blood may undoubtedly be effused into the air-cells, and yet these organs may recover perfectly their functions. But if abundant hæmoptysis occur, it is sometimes rapidly fatal. The mode of treating these cases is the same as in cerebral congestion. Your chief remedies are blood-letting, general or local, purgatives, and external stimulants. In cases in which a sudden effusion of serum has taken place into the air-cells, the same indications are to be fulfilled. Although, as a general rule, pulmonary œdema indicates a more advanced stage of the disease and less vital power than pulmonary apoplexy; the tendency is to dropsy or to passive congestion, rather than to hemorrhage—the congestion is of a less active character.

Bronchitis, in a slight degree, accompanies almost every case of enlargement of the heart. When trifling, it is perhaps best not to interfere with it. But such patients are subject to attacks of acute bronchitis, which aggravate the dyspnœa to such a degree as to constitute a form of suffocative catarrh. A prolonged and distressing cough with abundant expectoration, the sibilant rhonchus over the chest, with perhaps a mucous rattle at the base of the lungs, indicate the nature of the complication. It may even prove the immediate cause of death frequently, by passing into pneumonia. Such cases do not indicate general bleeding, unless the dyspnœa is very urgent and there is a considerable vigor of constitution. Cups to the chest, sometimes dry cupping, are very beneficial; also purgatives, external irritation, the use of antimony or of ipecac, in the early stage, to be followed by the use of stimulating expectorants—these are the best means of relieving the symptoms.

When enlargement of the heart has advanced so far that there is no longer any hope of relieving the internal congestions, you must still attempt to relieve the distress that torments the unhappy patient. Opium will sometimes enable him to procure an unquiet sleep, and other sedatives may be employed with the same object. Mild purgatives will relieve the abdominal distension; bandages applied to the legs are also useful means of relieving the distension of the extremities. Sometimes you will be obliged to resort to puncturing the limbs to relieve the distension; but this should always be done with great care, inasmuch as erysipelas may follow its injudicious employment.

In the treatment of the inorganic, the mere functional disturbances of the heart, both local and general remedies addressed to the heart are of no use. The indication is, after finding out the real source of trouble in some remote organ, or in the general condition of the system, or in some peculiar habit, to endeavor to remove it by appropriate means. These means are so various in different cases, and are so unconnected with the treatment of real disease of the heart, that I shall not attempt to enter upon their consideration. I will, however, offer this suggestion. A treatment directly opposed to that indicated in enlargement of the heart, is generally the most useful. Active

exercise, tonics, are often indicated. Thus the means which relieve one class of affections will aggravate the other. To treat a case of chlorosis, of dyspepsia, of exhaustion from the habit of masturbation, as if real disease of the heart was present, is a sad mistake, yet not a very uncommon one. The aggravation of the symptoms by improper treatment, will often lead the practitioner to suspect an error in the diagnosis. But let me repeat, in conclusion, what I have strongly insisted upon in my former remarks, that the best source of diagnosis is in the physical condition of the heart, in ascertaining whether any signs of enlargement of that organ, or of valvular disease, exist.

LECTURE XXXIII.

MISCELLANEOUS DISEASES OF THE HEART.

Carditis, or inflammation of the muscular substance of the heart.—Abscess of the heart.—Induration.—Softening.—Fatty degeneration of the heart.—Rupture of the heart.—Atrophy of the heart.—Tubercles in the heart.—Dropsy of the pericardium: symptoms.—Air in the pericardium: symptoms.—Displacement and malformation of the heart.—Polypi of the heart: symptoms and treatment.

There are several morbid conditions of the heart, which are of rare occurrence, and are unattended by characteristic symptoms, which require at least a cursory notice; and which, although entirely unlike in their nature, I shall introduce to your notice in the present lecture.

In considering the inflammatory affections of the heart, I have spoken only of endocarditis and of pericarditis. *Is there such a disease as carditis*, or simple inflammation of the muscular substance of the heart? Laennec observes, that there is not, perhaps, a single well-established case on record of general carditis, either acute or chronic in its character. This opinion, although now known not to be absolutely true, is yet so near the truth, that I have been unable to find more than two cases

which may properly be classed in this category. A case is alluded to by Dr. Latham, in the London Medical Gazette, in which the patient died after two days' illness. On post-mortem examination, the whole heart was found of a dark-red color, with its substance softened, and with innumerable points of pus oozing from among the muscular fibres. The other case is recorded by Bouillaud, and occurred in the practice of Dr. Simonet. The patient was about sixty years of age, and the attack occurred during the course of a rheumatism. The action of the heart was tumultuous, and there was great dyspnœa. A number of small abscesses were found in the muscular substance of the heart, which was of a yellowish-gray color, and was softened.

Most of the cases of inflammation of the muscular substance of the heart have been found in connection with inflammation of the membranes, but they are not numerous. Indeed, it is a well-established law in pathology, that inflammation of the serous membranes does not readily extend to the subjacent tissues, if we except the subserous cellular tissue, which, indeed, is the chief seat of the disease in serous inflammations. A very different law is observed in cases in which the substance of an organ is attacked. In this case, the inflammation almost always extends to the serous tissues, and is marked principally by the exudation of lymph. This is, no doubt, one reason why muscular carditis is so very rare. Yet, when the inflammation of the muscular structure is extensive, and the serous inflammation only marked by a moderate effusion of lymph, the disease may be properly called carditis. It would seem that the secretion of pus in cases of pericarditis, are more apt to be followed by carditis. Corvisart gives a striking example, in which the heart was softened, and of a pale-yellow color, with a good deal of vascular injection, and with a fatty-looking substance, probably lymph, or concrete pus, existing among the muscular fibres.

The existence of a circumscribed abscess in the muscular substance of the heart, has also been occasionally noticed, and these abscesses may open either externally into the pericardium, if adhesions do not form, or internally into the cavities of the heart, which is more common. I have seen one instance of this. It is

muscular substance of the left ventricle was petrified, so to speak, by numerous small bone-like masses, and accompanied by an increased development of the parts affected. These changes are also, probably, changes in the lymph which has originally been deposited in the muscular substance of the heart.

Softening of the heart sometimes occurs, also as a consequence of inflammation. The color of the organ is of a deep red, or livid or brownish tint, and a deposit of lymph or of pus may be present. But I am not disposed to regard even a majority of the cases of softening of the heart as dependent upon inflammatory action. In patients who have died of typhus fever, it is not uncommon to find the heart softened and of a livid redness—a change produced by a partial decomposition even during life. I have also observed in cases of enlargement of the heart a partial tendency to softening of the muscular substance, and I have been disposed to refer it to a perverted nutrition, not to inflammation.

The symptoms of softening of the muscular substance of the heart are easily understood. The impulse becomes feeble, the heart may flutter and beat irregularly, and the pulse may assume the same characters. Dyspnœa is also a prominent symptom, and sometimes a tendency to fainting, or even angina, and according to Dr. Hope, there is a strong tendency to pulmonary apoplexy. I am disposed to believe that the feeble and irregular action of the heart, noticed in many advanced cases of enlargement of the organ, with increased dyspnœa and obstinate dropsical effusion, are owing, in part at least, to a tendency to softening of the muscular substance. This condition may, however, terminate life suddenly by rupture of the heart.*

* Dr. Hope has remarked in the last edition of his work on the Heart, that a short, flapping, systolic sound, resembling the second or diastolic sound of the heart in its tone, attends a softening of the heart. This is also noticed in dilatation of the heart. Dr. Stokes, of Dublin, has also studied the action and the sounds of the heart in the tendency to softening of the organ, which occurs not unfrequently in the course of typhus fever. He regards this condition as indicated when there is a diminution, or a cessation of impulse, a proportionate diminution of both sounds of the heart, or a preponderance of the second sound.

There is, upon the table, a specimen of softening of the heart, with rupture of the left ventricle near the apex, which illustrates this form of disease. The specimen was presented to me by Dr. Lasher, who also favored me with the following particulars of the case. The patient was a retired sea-captain, seventy-three years of age, a free liver, but not intemperate, and extremely corpulent. He had previously presented some symptoms of cardiac disease, and he had an irregular pulse. The day of his death he had dined very heartily, and walked four miles immediately after dinner, and became much fatigued. He was attacked, during the afternoon, with sudden and severe pain in the epigastrium, and with a disposition to vomit. When first examined, the pulse was regular and of good strength. The case was regarded as an attack of colic, and an emetic was prescribed. But the pain continued, moving up a little higher to the precordial region, the pulse became feeble and irregular, and the patient began to sink. He died suddenly, while raising himself in bed, about twelve hours after the attack.

The post-mortem examination was made the next day, the weather being clear and cool, in the month of April. The heart was generally enlarged and flaccid, and of a pale slate-color. Although removed within twenty-four hours after death, evident decomposition had commenced. Near the apex of the left ventricle, a portion, about an inch and a half in diameter, was of a brownish-red color, very soft and broken down, and this condition extended quite through the muscular substance. The pericardium covering this portion was penetrated by three or four apertures large enough to admit a small pea, and which had more the appearance of ulceration than of rupture. Yet there were no marks of inflammation in the pericardium, or in any other portion of the heart. The pericardium was filled with blood; numerous atheromatous and ossific deposits were found upon the aortic valves and in the course of the aorta.

The treatment of softening of the heart is tonic and supporting. In cases in which the tendency to softening is dependent upon typhus fever, stimulants are indicated, as wine, brandy, ammonia. When the general system is cachectic, or when anemia exists, tonics, especially the preparations of iron, are the

best remedies. The diet should be simple and nutritious. Passive exercise should be enjoined, but the greatest care should be taken to guard against sudden mental emotion or strong physical effort.

It is highly probable that many cases which have been described by pathologists as softening of the heart, in which the organ was found soft and flaccid, pale, or of a dead-yellowish tint, were, in fact, cases of a different affection, now known as the *fatty degeneration* of the heart. In this morbid change, the contents within the sheath of the muscular fibre are changed to fatty matter, either by being converted into fat, as some think, or by a deposit of fatty matter taking the place of the absorbed muscular elements.*

* Dr. Quain, in his valuable paper on fatty diseases of the heart, published in volume xxxiii. of the Medico-Chirurgical Transactions, and to which I am indebted for many important facts, thus describes the microscopic appearances of the disease: "When a portion of the heart thus diseased is examined, the first thing that meets the eye is a want of the transverse striæ which mark the fibres of all the voluntary muscles, and less distinctly those of the heart amongst the involuntary muscles. When a portion of the object in which the disease has but commenced is examined, it will be observed that in addition to the faintness with which the transverse crossings are seen, a number of small dark dots stud the fibres in many points, evidently situated within the fibre. A few such dottings do not appear to be inconsistent with a tolerably healthy state of the fibre. * * * As the observer, however, approaches the seat of more decided disease, he finds that the cross-markings have, in many points, disappeared, and that the black dots have increased in size and number: some of the dots are now found to be transparent in their centre, and to assume some order in their arrangement. Sometimes the centre of a fibre is occupied by a long row of opake points, forming a continuous line in the course of the filament, sometimes two or three lines lie side by side, the dark lines being broken by transparent apertures or circles. Less frequently the lines lie transversely to the fibre. Thus may be traced, as Mr. Paget has described, the change from the particle of molecular fat, the black dot in the fibre, to the large oil globule, with its transparent centre. * * * This fat, be it remembered, is not in the ordinary fat-cells, such as are found on the surface of the heart or among the fibres. The fat-globules placed within the fibre are much smaller, appear to have a mere albuminous envelope, and are extremely like the oil globules of milk. They often escape from the broken fibres, float as free oil globules, or lodge between the fibres. * * * The size of the globules of fatty matter rarely exceeds that of the blood corpuscles; that of the black dots may be, in the first instance, not more than one-tenth of this measurement. That these particles and globules are composed of fat, is demon-

The cases which Laennec described as softening, with a change of color compared to that of a dead leaf, were probably cases of this fatty degeneration. The disease, even when extensive, does not, according to Quain, affect equally every portion of the organ, so that its surface appears marbled with pale-red and yellow, and in different degrees, the yellow tint predominating where the degeneration is most advanced. The consistence of the heart also is diminished in different degrees in different cases. Sometimes it is torn with the greatest facility, and the torn surface may present a distinctly granular appearance. It often exists (twenty-three times in thirty-three cases, Quain) with enlargement of the heart. But, in some cases, the heart has been found to be smaller even than natural.*

The influence of this condition upon the heart, and upon the system generally, is easily understood. The power of the heart is weakened, and the consequences of obstructed circulation ensue. The symptoms of softening of the heart are those of this fatty degeneration: dyspnœa easily excited; a tendency to fainting; coma; precordial pain; angina pectoris; a feeble pulse; dropsical effusions; and very frequently sudden death, often from rupture of the heart. The impulse of the heart is feeble, with or without the physical signs of enlargement of the organ.

The conditions which seem to favor this affection of the heart are such as tend to impair the nutrition of the organ. It has frequently been noticed in connection with different exhausting diseases, and it is still more intimately connected with local causes which tend to produce the same result. Thus it has been frequently found in connection with obstruction of the coronary arteries from the atheromatous deposit, a form of disease which may also be regarded as connected with a fatty degeneration, and which I shall describe more particularly when I speak of aneurism

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strated by their highly refractive properties, and by the action of ether upon them when they have escaped from the sarcolemma."

* According to Dr. Quain, all parts of the heart's muscle are liable to this change, but not equally so. First in frequency is the left ventricle, then the right ventricle, then the right auricle, and least of all, the left auricle. It attacks the columnæ carneæ and the inner layers of the muscular fibre most distinctly.

of the thoracic aorta. The pale, muscular substance, which is sometimes found under the thick, false membranes from old pericardial inflammation, may be the result of the same change, caused by imperfect nutrition from long-continued pressure. The disease also occurs most frequently in those advanced in life; is more common in males than in females. In some cases there is a general tendency to the deposit of fat in the system, but this tendency is not very remarkable. Mr. Canton, a late English writer, has remarked that the arcus senilis around the cornea, and which is also a fatty degeneration, has usually associated with it fatty disease of the heart.

The deposit of fat upon the heart, especially under the pericardium, is a kindred affection. It is not unfrequently associated with the fatty degeneration, but differs from it in some important particulars. It is more frequently associated with a general tendency to the deposit of fat in the system, although Bizot has shown that it frequently attends the progress of emaciation in tuberculous phthisis, especially in women, in whom it often is associated with the fatty liver. It is, like the deposit of fat generally, a deposit of fatty tissue composed of large vesicles. (Plate II. Fig. 13.) It attacks the superficial portions of the heart more distinctly than the internal portions. Indeed, it generally makes its first appearance along the track of the coronary vessels, first showing itself in the fissure between the right auricle and ventricle, extending along the vessels to the apex, and finally spreading over the surface, particularly of the right ventricle. But it may penetrate more deeply. It insinuates itself between the muscular fibres, which become scattered, atrophied, until the muscular substance of the heart is weakened, even, perhaps, to the point of rupture. I have seen the deposit of distinct masses of fat even between the layers of endocardial membrane that forms the mitral valve.

The conditions under which this form of fatty disease takes place are not as yet well understood. But being associated often with a general tendency to corpulency, it may be regarded, like that tendency, as a rather deteriorating process. It is a common popular error to associate a tendency to corpulency with a condition of health. It is not an evidence of vigorous health.

Corpulent persons are usually deficient in both mental and physical energy, and that independently of the unnecessary weight they are obliged to carry with them. They are very different from the "bone and sinew" men, so well illustrated in our backwoodsmen, whose endurance of fatigue and mental energy are so remarkable.

The symptoms which attend this deposit upon the heart are not marked, unless it amounts to a disease of structure, by compressing the organ and attenuating the muscular fibre. Its symptoms are then those of a weak heart—symptoms which I have already indicated in speaking of softening of the heart, and of the fatty degeneration of the organ. The same principles of treatment which I alluded to when speaking of a tendency to softening of the heart, should guide you when you have reason to suspect that a tendency to fatty disease of the heart exists.

Rupture of the heart is an occasional cause of sudden death in heart disease. It may occur from softening of the heart, as in the case which I have related; but it occurs, probably, more frequently in connection with fatty disease of the organ. Mr. Quain states, that it occurred in eight of thirty-three cases of fatty degeneration, which he has analyzed; and in three of fifteen cases of fatty growth upon the heart. But sudden death may occur still more frequently in cases in which the heart is weak, from syncope. Rupture of the heart is generally complete, and death soon follows, but it may happen, especially in the fatty degeneration of the organ, that the internal fibres of the heart alone give way, and thus a small cavity may be formed containing a clot of blood, and communicating with one of the cavities of the heart, until, finally, a false aneurism of the heart itself is developed.

Rupture of the heart from wounds of the organ belong rather to surgery than to medicine. It is generally supposed that almost instantaneous death follows this accident. But this is not always the case. I recollect the case of a young sailor who was stabbed through the left ventricle and the aorta near its origin with a knife. He ran a considerable distance after the accident,

and lived ten or fifteen minutes. The pericardium and the left pleural cavity were filled with blood. There is a specimen of extensive laceration of a ventricle in the museum of the Hospital, caused by the crushing of the body between a vessel and the wharf. The liver was also lacerated, yet the patient, a young sailor, lived about thirty-six hours.

I have also seen a case of rupture of the pericardium from a blow, without material injury to the heart.

A sailor of middle age, a stout, healthy man, was struck in the breast by a bale of goods swinging against him, as he was discharging a cargo, and by which he was knocked down the hatchway. He was brought into the Hospital paralyzed in both upper and lower extremities, but in the full possession of his senses. His skin was warm, his pulse good and regular. He lived forty-eight hours. On examination after death, there was a fracture of one of the cervical vertebræ, a laceration of the right lung near the summit, and a corresponding fracture of the second and third ribs in front. There was also a comminuted fracture of the sternum over the precordial region. The pericardium was lacerated, from above downward, over the surface of the right ventricle, to nearly its whole length (four inches). On the surface of the ventricle there was a spot as large as a sixpence, looking like a bruise. But very little blood was effused into the pericardial sac. The heart was natural. A small quantity of blood was found in one of the pleural cavities.

Atrophy of the heart sometimes occurs, and, I believe, chiefly in connection with wasting chronic diseases. It has been ascertained by Bizot, that the hearts of patients who die of tuberculous phthisis, are smaller than the hearts of those who die of most other diseases, and it is probable that the atrophy would be still more marked, was it not for the obstruction of the circulation through the lungs. I suspect that the most marked cases of atrophy will be found to occur in connection with cancer of the stomach.

You may suspect that atrophy exists, when the action of the heart is feeble, its impulse small and limited, the sounds less

intense than natural, and when unusual clearness on percussion over the precordial region exists.

Atrophy of the heart, but with very different physical signs, has been noticed in cases of old and thick false membranes covering the heart, also after long-continued and abundant liquid effusions. In both cases the atrophy is the result of pressure, and the symptoms and physical signs are those of chronic pericarditis.

Tubercles and *cancerous deposits* are sometimes found imbedded in the muscular substance of the heart, or existing in the false membranes of the pericardium. I have witnessed, I think, two cases in which tubercles existed in the heart, but in neither case have I any knowledge of the attending symptoms. I have never seen a case of cancer of the heart. In speaking of cancer of the mediastinum, I described a cancerous mass that was developed around the origin of the great vessels that spring from the heart, so as sensibly to compress these vessels. The symptoms were those of valvular disease of the heart; a very distinct blowing sound, with the first sound of the heart, existed at the base of the organ.

I will simply allude to the fact, that *hydatids*, *serous cysts*, and *apoplectic effusions*, have been found in the substance of the heart. I have seen a case of the latter disease. The history and the symptoms of these organic changes are unknown.

Dropsy of the pericardium is, in my experience, a very rare form of disease. I have never seen it, except in connection with enlargement of the heart, and with effusion into other serous cavities. My observation agrees entirely with that of Laennec, that when a considerable effusion of serum exists in the pericardium, it exists at the same time, and still more abundantly, in other cavities. Authors speak of an idiopathic effusion into the pericardium, a simple excess of the serous secretion from irritation, but I have been unable to find a single recorded case. There are, indeed, cases of pericarditis in which the effusion of serum is abundant, while that of lymph is very trifling, but these cases evidently belong to a subacute, or chronic inflammation of the pericardium.

I have stated that dropsy of the pericardium is a rare form of disease, and yet there is a prevalent opinion among practitioners that it is of common occurrence. I have, not unfrequently, in consultation with those who are not in the habit of practising auscultation, heard the opinion expressed, that dropsy of the pericardium existed, and yet, in a careful examination of the case, nothing of the kind could be detected. This has most frequently happened in cases in which the disease has become suddenly aggravated, with a tendency to anasarca of the lower extremities. It is true that you will often meet with a moderate effusion of serum into the pericardium after death. It is not uncommon to find one or two ounces in this cavity, especially after a prolonged agony from any cause. But as this quantity, or even a larger quantity, is not unfrequently found in persons dying of various diseases, it cannot be regarded as a pathological phenomenon, but must be looked upon as an affair of the agony, or as occurring even after death. Corvisart states, that an accumulation of even six ounces is not to be regarded as an evidence of disease.

The symptoms of dropsy of the pericardium are, therefore, those of enlargement of the heart. There can be no doubt that a considerable effusion will aggravate the symptoms already existing, increasing especially the oppression and dyspnœa, and perhaps inducing an irregular pulse in some instances. The existence of serous effusion in other cavities renders dropsy of the pericardium a possible complication, but not, in my experience, a very probable one.

You can only be sure that effusion into the pericardium exists by the study of the physical signs, which are precisely those of pericarditis with liquid effusion. The heart is pushed backward from the anterior parietes of the chest, towards the spine; consequently, the impulse of the heart is diminished; and if the effusion is very great, it may be entirely absent, at least to the touch. The sounds of the heart also become distant and obscure. It is stated on the authority of Corvisart—but I have never noticed the fact—that in cases of considerable effusion, the maximum of impulse in the heart changes its position from one moment to another, as the heart moves about from right to

left in the fluid. It is also stated by Dr. Hope, that the impulse is fluctuating. This also I have never noticed, and the general impression among auscultators seems to be, that it is of rare occurrence. Percussion is an important aid in these cases. The dulness over the precordial region is much increased in degree as well as in extent, reaching sometimes as high as the second rib, and extending laterally even beyond the nipple. The area of dulness is sometimes conical in its shape, with the apex of the cone upward; and on post-mortem examination, you may find this to be the shape of the distended sac. In a case of chronic pericarditis, in which the liquid effusion was enormous, the dulness was noticed to recede an inch or more from the left side, and to increase to the same extent on the right side, as the patient turned from the left to the right side, and vice versa. This will, probably, also be sometimes observed in cases of great dropsical effusion. There may also be noticed a dilatation of the precordial region, and an absence of the respiratory murmur.

It is a question how much fluid must exist in the pericardial sac to become evident by a physical examination. Laennec states that a pint is necessary; but with the improved means of physical examination, it is probable that a less quantity could be easily detected. In a case that occurred to me some time ago, the quantity during life was, probably, less than this. The patient was a seaman, thirty-two years of age, who entered the Hospital with the symptoms of enlargement of the heart. He had been attacked with acute rheumatism six years before, and his cardiac symptoms had been noticed about a year before. He had hydrothorax on the right side, and anasarca; the pulse was accelerated, but regular. On examining the precordial region, the whole anterior portion of the left side of the chest appeared more prominent than the right side. The dulness on percussion extended an inch above and the same distance outside each nipple. On applying the hand to the precordial region, the action of the heart could not be felt, even at the apex. No respiratory murmur was perceived over the body of the ventricles. The patient lived nearly four months after this, the tendency to dropsical effusion gradually increasing, and died after a prolonged agony. The pericardium was found much dis-

tended with fluid; it contained at least a pint of serum. No traces of pericarditis were noticed.

The treatment of dropsy of the pericardium is that of other dropsical effusions dependent on obstructed circulation from disease of the heart. But as it generally indicates a higher degree of obstruction than even other serous effusions, it is, of course, more difficult to remove. I have succeeded in removing it, or in materially diminishing its quantity by mercury, combined with squills and with digitalis.

It has been proposed to puncture the pericardium and draw off the fluid, in cases in which the accumulation is considerable and obstinate. Dessault attempted this twice, but in both instances he entered the cavity of the left pleura, for it was there that the fluid existed. The diagnosis of such cases was very uncertain in the time of Dessault. In the present state of knowledge it might easily be done, either by a trocar passed between the cartilages of the fourth and fifth ribs, on the left side near the sternum, or by trepanning the lower portion of the sternum, as Laennec has suggested. But the question is, Would it do any good? I must confess, that some years ago I should have been in favor of attempting it in certain cases; but now experience and reflection have brought me to a different conclusion. As the disease in a majority of cases, if not always, exists as a complication of enlarged heart, and is dependent on an incurable obstruction to the circulation, either one of two things must infallibly happen. The pericardial sac will rapidly fill again with fluid; or inflammation will ensue, and adhesions will form, which will aggravate the previous disease of the heart more than the dropsical effusion had done. Indeed, there is but one case in which I would think of tapping the pericardium. If I supposed that a considerable effusion of pus or of seropurulent fluid existed in the pericardium as a consequence of pericarditis, and other means had failed to remove it, I would then resort to tapping. The operation itself cannot be difficult; it is the consequences that are more serious. The admission of air into the cavity is, perhaps, unavoidable, and unless this cavity contains a purulent fluid, inflammatory reaction is pretty sure to follow.

Laennec states that it is not uncommon to find gas in the pericardium after death, especially in those who have died after a prolonged agony. Indeed, he seems to think that it is not an uncommon thing for air, in moderate quantities, to be secreted during life, and to be rapidly absorbed again. He entertains a fanciful idea in explaining the occasional fact noticed principally in nervous persons, that the action of the heart can be heard at a considerable distance from the individual, by the existence of air in the pericardium. I was surprised to find this accumulation of air so common in the experience of so accurate an observer, when I had never met with it in hardly a single instance. Still, there can be no doubt that air is sometimes secreted into the pericardium, especially in cases in which a purulent secretion has previously existed, and from which the gas has probably been eliminated. In these cases, the sound of fluctuation has been heard with the impulse of the heart, and it has been compared to the dashing sound produced by a water-wheel. In a case in which gas alone was secreted into the pericardium, I should expect to find an unusual clearness on percussion over the precordial region, while the other physical signs would probably be identical with those of liquid effusion. If liquid and gas existed together, the dulness would vary with the proportions of each, and its precise seat might be made to vary, by altering the position of the body. The dashing sound, synchronous with the action of the heart, might also exist.

I have met with a single case in which I supposed that air existed in the pericardial sac, in connection probably with a slight serous effusion. I will relate it.

A young gentleman, about twenty years of age, and occasionally subject to slight muscular rheumatism, but in the habitual enjoyment of excellent health, was attacked suddenly while he was walking rapidly, with pain in the middle of the back, which soon passed forward to the precordial region. The breathing became exceedingly oppressed, so as to compel him to stop. On the day of the attack, a sound in connection with the action of the heart was noticed in the precordial region when the patient lay on the left side. In any other position of the body it was not heard. It was heard even at a distance

of three feet from the body. The pulse was at one time fifty in a minute; at another examination, it was natural in frequency. It was occasionally intermittent. There was no fever.

I saw the patient on the fourth day after the attack. The pain in the precordia had continued two days; it then subsided. The dyspnœa also diminished, but still existed in a slight degree. There was no pain in the precordial region. The sound on percussion over this region was remarkably clear, more so than over the corresponding portion of the right side, and resembled somewhat the cracked-pot sound. Whenever the patient lay on the left side, a double, gurgling or splashing sound was heard, having its maximum just above the apex, and to the right side, synchronous with the sounds of the heart; but which latter sounds, with attention, could be distinguished from the splashing sounds. The first sound of the heart in particular was very clear. The impulse of the heart was strong and superficial, and on placing the hand just above, and to the right of the apex, a gurgling could be felt over a small space; but this as well as the sound ceased immediately when the patient assumed any other position than that on the left side. When, on the contrary, this position was observed, the sound had always been heard, even by the patient, during the period of four days since its first occurrence. I heard it distinctly when standing at least two feet from the patient. The pulse was natural—there was no febrile excitement. Indeed, with the exception of a slight dyspnœa and the morbid sounds, the patient was perfectly well. He was brought to me from out of the city, and I have never heard from him since.

Displacements of the heart have already been alluded to, so far as they are connected with disease of other organs. The principal causes of displacements are liquid effusion into the cavities of the pleura, particularly of the left pleura, the development of tumors in the chest, and the enlargement of different organs, either in the chest or abdomen. There are many curious cases on record of congenital displacement of the heart, from hernia through the diaphragm, or through the parietes of the chest and abdomen. Bouillaud relates a remarkable case of

general displacement of the viscera of the thorax and abdomen. The heart was not merely displaced as a whole, from the left to the right side of the thorax, but its different portions were also transposed. Thus, the right auricle and ventricle was on the left side of the heart, and the left auricle and ventricle on the right side.

The valves of the heart are also sometimes deficient, or in excess. I think I have met with a case in which but two sigmoid valves existed, and another case in which there were four. I have also met with a case, alluded to in a former lecture, of congenital contraction of the mitral orifice, in an individual who lived to the age of twenty-three years. I have also spoken of the communication of the two sides of the heart through the foramen ovale; from an imperfection in the septum of the ventricles, and from the aorta and pulmonary artery having a common origin from the two ventricles, in describing the disease known as cyanosis. Most of these morbid conditions are congenital, although there is reason to think that the communication between the ventricles may be the result of ulceration or abscess, and perhaps also the foramen ovale, when once closed, may be torn open again by violent action of the heart. It is not uncommon, however, to find this foramen open without any apparent disturbance in the functions of the heart.

Finally, in certain monsters with two heads and with two necks, two hearts have been found. A case is recorded in which three ventricles existed. While, on the other hand, especially in anencephalous monsters, no heart, or only the rudiments of a heart have been noticed. An auricle or a ventricle, or both, have also been found wanting, also the pericardium.

The vena azygos has been seen to open into the right auricle, and the same termination has been noticed in the hepatic veins. The ductus arteriosus may continue open after birth, or may communicate directly with the right ventricle, and the pulmonary veins with the right auricle, and the vena cava with the left auricle.

I will conclude this lecture with some remarks on *polypi in*

the heart. I have already alluded to the subject on another occasion, but it is of sufficient importance to require a more attentive consideration.

Every one who has made but a limited number of post-mortem examinations, must have remarked the frequency with which clots of blood are found in the cavities of the heart, and especially in those of the right side of the heart. A cursory examination is enough to satisfy you that these clots resemble entirely those formed by the blood after standing in a vessel, when drawn from a vein in the arm. There is no doubt that the explanation of the fact is the same in both instances. It is the stasis of the blood in the heart, as in the vessel, which induces its coagulation—the last act of its vitality. These clots are soft, and easily torn. They do not adhere to the walls of the heart, they contain a large proportion of coloring matter, although not an equal proportion in every part, and after filling, more or less, the cavities of the organ, often extend into the great vessels which spring from the heart. These clots are evidently formed during the agony that precedes dissolution, or, perhaps, even after the heart has ceased to beat. Polypi in the heart are a step beyond this simple coagulation. If you beat the blood for a considerable time in a vessel with small twigs, you will gradually separate the fibrine. It appears of a yellowish-white color, it is elastic and tenacious. If the blood stagnates partially in the heart, from an obstruction to the orifices of the heart, the organ still continuing its action, the fibrine is separated in the same way, and presents the same appearances and conditions. In the course of its formation it becomes entangled in the meshes of the tendinous chords and fleshy columns, and finally presents traces of organization, and adheres firmly by cellular tissue to the lining membrane of the heart. It is probable that fibrine thus separated from the blood by stasis and by agitation, is capable of organization, like the lymph effused from an inflamed surface. This, however, is not the only way in which these polypi are formed. If you look at an ordinary case of phlebitis, you will find that portion of the vein in which the inflammation exists, filled by a clot, soft and red, like an ordinary coagulum. Is this the result of mere stasis of the

blood, produced by a paralysis of that portion of the vein which is inflamed? I think not. There is no proof of paralysis, or of any other cause of obstructed circulation. Yet the clot forms, and at the point where the inflammation exists; and this clot becomes gradually white and firm, organized, and adherent to the lining membrane of the veins by cellular tissue. Does the inflammation of the vein itself dispose the blood in contact with it to coagulate and thus form a clot? There is reason to think that it does, but there is no positive proof. It is in this way, probably, that inflammation of the lining membrane of the heart predisposes to the formation of polypi, for they are very apt to exist in such cases. It does not appear to me that the stasis of the blood in the heart has much to do with the formation of these polypi in acute endocarditis, because there is, probably, not much obstruction to the circulation through the heart, until the polypi themselves produce it by filling up its cavities. There is, it is true, a thickening of the valves, which may impede somewhat the circulation, but then the action of the heart is increased, and more than enough to remedy this obstruction.

You may, then, recognize two distinct sources of polypi, stasis and inflammation. It is probable that something more is necessary, in certain cases, for it is certainly true that polypi do not form in every case of endocarditis, and you will as certainly meet with cases of long-continued obstruction to the circulation through the heart, also, without their formation. There can be no doubt that these two causes are united in many cases, and it is also reasonable to believe, that an accidental increase of the proportion of fibrine in the blood, from inflammation of some remote organ, may predispose to the formation of polypi in the heart. It has been observed, that polypi are especially apt to form in the heart in the course of a pneumonia; and I have observed, repeatedly, in this disease, that the pulmonary vessels were filled with fibrine, in other words, with polypi. Several interesting questions might be asked in this connection. Do these clots in the pulmonary vessels predispose to the formation of similar clots in the heart by obstructing the circulation; or by an extension of inflammation from the vessels to the heart; or

is the increase of fibrine in the blood generally, from inflammation, the true cause? Each and all these causes may operate, in certain cases, to produce polypi in the heart.

There is another view of the inflammatory origin of these polypi which has been taken by some writers. It is supposed that they can be formed by the direct exudation of lymph from an inflamed endocardium. There is no doubt that lymph is thus effused; the vegetations and patches in the heart prove this. Probably this effused lymph may unite with the fibrine separated from the blood in the heart, and increase the size of the polypus and the rapidity of its formation. Indeed, it seems to me that certain small globular polypi, attached to the heart by a pedicle, may be formed entirely in this manner.

Pus is said to be sometimes found in the centre of these polypi. The question is, how does it get there? Is it the result of inflammation, attacking the organized polypus, or is the pus secreted by the lining membrane of the heart, or transported there from the veins, and afterwards inclosed in a coagulum of fibrine, from the coagulation of the blood in the cavities of the heart? Both these opinions are maintained; neither are proved to be true. It is more probable, I think, that small masses of fibrine, in various stages of decomposition, are found in these polypi, and have been mistaken for pus.

Cardiac polypi are usually elongated, with one extremity twisted among the tendinous chords or muscular columns of the heart, and sometimes firmly adherent to the lining membrane by cellular tissue. The extremity that is loose, lies in one of the cavities of the heart, or extends into an orifice. Sometimes these bodies are globular, and attached by a pedicle to the walls of the heart. They are more white, more firm, more fibrous in their texture, the older they are—in a word, they are better organized; but not always. Sometimes they become friable from the disorganization of the fibrine. It has been thought that many even undergo changes of a cancerous nature, but this is simply possible.

The symptoms of polypi are those of mechanical obstruction to the circulation, and these will vary with the rapidity of their formation. In cases of endocarditis, in which, perhaps, they

form most rapidly, you will notice great dyspnœa and general distress, syncope, venous congestion, a tendency to coma, a small pulse, and coldness of the extremities, as the symptoms most attributable to the formation of polypi. If, in cases of chronic disease of the heart, and especially of the orifices, you find the dyspnœa suddenly increased, and other prominent indications of increased obstruction to the circulation, and no other cause for this be noticed, you may suspect the formation of a polypus in the heart.

I do not think, however, that you would be justified in entertaining more than a suspicion. I know of no certain signs of the presence of polypi. They may induce dyspnœa and precordial pain and anxiety; they may, by partially obstructing the orifices of the heart, induce a blowing sound in some of its modifications; they may induce a small and feeble pulse: but these symptoms may all exist without their formation.

Polypi often form slowly, producing the effect of gradually obstructing the circulation of the heart, and in this there is nothing characteristic. But they may be the cause of sudden death. Louis, I think, mentions a case in which a polypus became, as it were, doubled upon itself, and thus filled up an orifice, causing sudden death. I have also seen a globular polypus capable of filling half the right auricle, which fell upon the auriculo-ventricular orifice so as to close it entirely. The patient, in attempting to close a window, suddenly expired. It is probable that this polypus was originally attached to the lining membrane of the auricle by a pedicle which had suddenly been ruptured.

These polypi when formed suddenly, especially in endocarditis, require an active antiphlogistic treatment, both to diminish the labor of the heart, and to overcome the inflammation which causes their formation. It is possible, that when recent, they may again be restored to the mass of circulating blood, and no ill effects follow their formation. A small and indistinct pulse, a tendency to fainting even, is no indication against free venesection in these cases; for the pulse will rise and the strength improve with its use. It is probably true, as Bouillaud observes, that practitioners have sometimes abstained from free depletion,

and have even used stimuli, mistaking a false weakness induced by polypi in the heart.

In cases in which polypi have formed more slowly, and have been more or less organized and adherent to the walls of the heart, you cannot expect to remove them; indeed, you can have no certain evidence of their existence. The evidence is even less certain than in those cases in which they are formed suddenly and in connection with inflammatory action. But if you did know that an old polypus existed in the heart, you could do nothing beyond adopting those general indications for relieving an obstructed circulation, which I have mentioned when speaking of the treatment of enlargement of the heart.

I have recently met with a case in which a polypus induced no other symptom than intense dyspnœa, and in which no physical sign of its existence could be detected, although its presence was suspected during life. A gentleman of delicate constitution, aged about thirty-five years, was first attacked, eighteen months before his death, with a latent pleurisy, which was followed by an attack of tubercles in the summit of each lung. The pleuritic effusion was slowly absorbed. After passing the winter in a warm climate, he returned much improved in health, and continued so during the ensuing summer. But as the cool weather of autumn set in, he became worse. He coughed more, expectorated more, and his dyspnœa increased. His strength failed, and night-sweats ensued. About four days before his death, the dyspnœa became rapidly intense: he could not move without inducing a most aggravated paroxysm; and, although the weather was quite clear and cold, he required that all the windows and doors in the room should be kept open. I examined the heart carefully. Its action was rather tumultuous, but not in a marked degree; its rhythm was regular, and the natural sounds were heard distinctly, and nothing else; there was no dulness over the precordial region; the pulse was regular (about 120 in a minute) and of moderate force. He died at length, rather quietly, after continuing in a partially stupid state for some time. On examination after death, the left lung was found universally adherent, by old adhesions; it was diminished in size: the right lung also adhered by much more recent adhe-

sions. Both lungs, the right perhaps more than the left, were crowded in every part with small miliary tubercles; a few small and old cavities existed in the summit of each lung; the heart was rather large; recent clots existed in the left cavities, and also in the right cavities; but in the right ventricle there was a polypus, white, firm, and elastic, extending into the pulmonary orifice, and half filling it.

LECTURE XXXIV.

ANEURISM OF THE THORACIC AORTA.

Medical anatomy of this vessel.—Organic changes which lead to the formation of aneurism.—Different forms of aneurism.—Aneurism by dilatation, or true aneurism; false, or saculated aneurism; mixed aneurism; dissecting aneurism.

The thoracic aorta, like other arteries, is composed of three distinct coats. The internal, which is a continuation of the lining membrane of the heart, is an extremely delicate, shining, and transparent membrane, closely attached to the subjacent coat by a fine cellular tissue. It resembles, in appearance, the ordinary serous tissue, and the tendency it exhibits, when inflamed, to the effusion of lymph, allies it closely to this tissue in a pathological point of view. Beneath this, is the middle coat, which has been called the muscular coat, or the elastic coat, but which, in fact, is a compound tissue, composed of yellow elastic tissue, and non-striated muscular fibre. The third coat is formed of condensed cellular tissue, which has no distinct limits externally, but becoming gradually more lax, unites the artery to the surrounding organs. The principal strength of the artery is in the middle coat, which unfortunately is the most frequent seat of disease. In addition to its elasticity, it is capable of considerable distension, which is not the case with the internal or serous coat. It is the external coat, however, which possesses this capacity for distension in the greatest degree. These facts should be carefully remembered in the study of aneurism.

There is another fact in the anatomy of the thoracic aorta which is of especial importance in the history of aneurism. The aorta, at its origin, and from two to three inches after it leaves the heart, is destitute of its external cellular coat on its anterior and lateral portions, and its place is supplied by the much more delicate and inextensible serous tissue of the pericardium. In a male adult, in whom I recently made the examination, the distance occupied by the pericardium, upon the anterior surface of the aorta, was two inches and ten lines.

The medical anatomy of the thoracic aorta should be carefully studied in relation to aneurism. The ascending aorta is in contact, at its origin, anteriorly and to the right, with the right ventricle; posteriorly, with the two auricles; and to the left, with the pulmonary artery. As it emerges from the substance of the heart, it is in contact with the pericardium and with the pulmonary artery in front; to the left, it touches the left auricle; and to the right, the vena cava descendens, and the right auricle.

The arch of the aorta is in contact, posteriorly and to the left, with the left pleura, the phrenic nerve, and the par vagum; posteriorly and to the right, with the trachea, the left bronchus, the œsophagus, the thoracic duct, the recurrent nerve, the vertebral column, and numerous lymphatic ganglia. In its concavity lie the left recurrent nerve, the left bronchus, and numerous lymphatic ganglia. From its convexity spring the great vessels of the head and of the upper extremities, and just above it lies the vena innominata.

The highest point of the arch extends to about one inch from the top of the sternum in adults, and as high as the second dorsal vertebra posteriorly.

The descending thoracic aorta is in contact, anteriorly and superiorly, with the left pulmonary artery and veins, and below, with the pericardium and the œsophagus. Posteriorly it lies in front of the vertebral column, a little to the left of the median line, from which it is separated above by the thoracic duct, and to the right by the œsophagus, vena azygos, and thoracic duct. To the left lies the left pleura, and around are numerous lymphatic ganglia.

The calibre of the thoracic aorta, as well as the thickness of its walls, are not the same at all periods of life. This vessel, like the heart, increases in size and in thickness with the progress of age.*

The aorta may be regarded as an elastic tube, and any cause which tends to diminish this elasticity may lead to its dilatation, or to the formation of an aneurism. It is true that it is not uncommon to find the aorta somewhat dilated in cases of hypertrophy of the left ventricle, and then its walls are also usually thickened. Frequently there is disease enough in the vessel to explain what you may notice. Still it does occasionally happen that the increased force of the column of blood rushing into the artery may increase the calibre of the vessel, as well as the thickness of its walls, without any disease of its coats. Here of course the dilatation, as far as it extended, would be general. I have occasionally, however, met with a partial dilatation of the artery into a pouch, when I could detect no disease. In a great majority of cases, when you examine the aorta which is the seat of any form of aneurism, you will find abundant evidence of disease and an easy explanation of the appearances you witness. All the diseased appearances you will notice, of whatever nature, lead to one of two results: by destroying the elasticity of the coats, they lead to dilatation, or even to a cracking or fissuring, from the pressure of the column of blood; or else, by partially destroying the inner and the middle coat, they enable the impinging column of blood to distend the external coat into a sac, which sometimes attains an enormous size. But what are these diseased

* The accurate observations of Bizot indicate that the mean dimension of the ascending aorta is 31⅞ lines, in males, in the prime of life, while in advanced life it is at least ten lines greater. At the arch, just before the orifice of the left subclavian artery, the dimension in the adult, in the prime of life, is 25¼ lines, and in the descending thoracic aorta, about 23 lines.

The mean thickness of the coats on the ascending aorta, in the prime of life, is 0·76 of a line; while in old age, it is found to have increased to 0·80 of a line. The mean thickness of the coats in the prime of life, at the arch, is 0·78 of a line, and for the descending aorta, 0·65 of a line.

In females, the mean dimension of the vessel is about three lines less than in males, and the thickness is less, in about the same proportion, when compared with the thickness of the coats in the male.

conditions of the artery, and how do they explain the phenomena of aneurism?

I have already stated that, in a few cases, the coats of the artery appear to have simply lost their natural elasticity, and that the vessel has thus become dilated without apparent disease. The recent microscopic observations of Mr. Gulliver, and this statement has been confirmed by others, tend to show that actual disease does exist even in these cases, in the deposition of minute points of atheroma, invisible to the naked eye, and which must impair the natural elasticity of the middle coat. This atheromatous deposit, as Scarpa calls it, plays a very important part in the formation of aneurism. Its nature, as well as its original seat, has been a matter of much dispute. Some have supposed it to be a peculiar alteration of the middle coat, but recent investigations have shown that it is a peculiar deposit of fatty matter in the cellular tissue uniting the inner and middle coats. In its earliest stage it presents the appearance of small, yellowish, opake specks, scattered over the internal aspect of the vessel, which, by increasing in size and coalescing, form spots, and even large patches, sometimes rounded, sometimes irregular in shape and slightly elevated. The internal coat of the vessel, with the exception of this slight elevation, appears transparent and unaltered, while the middle coat is destroyed, to a greater or less depth, by the absorption induced by the pressure of the new deposit. In this stage of the disease no apparent changes are produced in the physical condition of the aorta, except a loss of elasticity and an unevenness of the internal surface proportioned to the extent of the deposit. But sooner or later new and far more important changes ensue. These yellow and opake patches, which have hitherto been firm and consistent, may begin to soften and to become more elevated. When pressed upon by the finger, they yield readily, and if the internal coat is ruptured, they discharge a yellowish matter sometimes resembling pus, but more frequently a grumous, friable, and pasty substance, resembling very much in appearance softened tubercle. This substance, if rubbed between the fingers, sometimes feels gritty, and is found to contain minute spangles of a silvery or yellowish hue, and which singularly enough, have been found to be crystals of

cholesterine. By this rupture of the internal coat, which may, of course, occur spontaneously, a small cavity resembling an ulcer is formed, having for its base the partially destroyed and disorganized middle coat, which is here friable, and yellowish in its hue. Another change noticed still more frequently in these atheromatous spots, very different in its progress, but leading ultimately to the same result—the partial destruction of the inner and middle coats of the artery, is their gradual conversion into a substance resembling bone, although differing from bone in its intimate structure and component parts. In the centre of the opake, yellow spot, a hard and somewhat transparent spec is developed, which gradually increases until it occupies the whole, or a considerable portion of its substance. Still it is covered by the transparent and unaltered internal coat. But gradually this coat is absorbed, and the bony plate lies in direct contact with the blood. It now acts as a foreign body, inflammation is excited about it, and it is thrown off into the current of the blood, leaving behind a circumscribed loss of substance by the partial destruction of the internal and the middle coats. Indeed, as in the first form of atheromatous degeneration, an appearance resembling ulceration is produced, having for its base the detritus of the middle coat. These two results of the atheromatous degeneration may be regarded as more closely connected than they appear to be at first sight. In the former, the deposit tends chiefly to a state of softening; but here I am inclined to think that the gritty particles may, possibly, sometimes be of the bone-like nature noticed more distinctly in the second form, and not always crystals of cholesterine; while the ultimate result, and this is by far the most important fact, is the same in both, viz., a complete destruction of the internal coat, and a partial loss of the middle coat, over a circumscribed space.

The atheromatous deposit appears to be entirely independent of inflammation in its origin, and often throughout its whole course; unless at the very last stage, and especially when the particles resembling bone are about to be cast off. It is rarely found in those who die young; it is exceedingly common in those who die at an advanced age. Its immediate causes are unknown, although, as I shall state directly, its deposition ap-

pears to be favored by the pre-existence of old inflammatory deposits. Like tubercle and cancer, it seems to be deposited under the influence of what is, vaguely enough, called a diathesis, and like these deposits, it seems to have a peculiar affection for a certain period of life, and to tend to the destruction of the parts affected.

Other changes in the coats of the aorta, leading to the formation of aneurism, are evidently inflammatory in their origin. The simplest of these changes is a wrinkled or shrivelled appearance of the internal aspect of the vessel. This is, no doubt, induced by a slight deposit of lymph in the cellular tissue, between the internal and the middle coats, and which, subsequently contracting, produces the wrinkling you will notice. This condition would naturally lead to a diminution of the calibre of the vessel, were it not accompanied by a loss of elasticity and firmness of the middle coat, which, as well as the internal coat, is commonly found irregularly thickened. Thus it is not unusual to find the vessel dilated by the force of the column of blood. Indeed, so brittle and inelastic do these coats become from these inflammatory changes, that they sometimes crack, and thus lead to the early rupture of the vessel—to one of the worst forms of aneurism.

Another inflammatory change often noticed, is the effusion of lymph upon the internal membrane of the artery. Sometimes this is so distinct as to be at once detected. But, in other cases, it is much more difficult of detection. I have frequently seen it spread out in a sheet over the internal surface of the artery so thin, smooth, and transparent, as at first entirely to escape notice. At other times it appears only in patches. This effusion of lymph is well worthy of a careful study, for it seems capable of organization, and of supplying the place of the internal coat when that is wanting. Thus, it may extend into an aneurismal sac formed by the external coat alone, and resemble the internal coat so completely as to be with difficulty distinguished from it. Nay more, it appears to undergo the same changes that are so frequently noticed in connection with this coat. Thus patches of lymph may be deposited upon its surface, and the atheromatous deposit may be formed beneath it.

The patches of lymph effused upon the internal membrane of the artery do not always preserve this delicate, transparent appearance. They frequently become much thicker and opake, of a milky or dead-white hue, resembling in appearance cartilage, although differing from it in structure. It is a disputed point, whether these cartilage-like plates pass into a state resembling bone. Bizot, who has carefully examined this subject, thinks that they do not: but that the atheromatous deposit is frequently formed under them, which by passing into bone-like matter and partially absorbing the cartilage-like plate above, might, without great care, be supposed to be a change in the latter deposit. Most pathologists, however, are of opinion that these cartilage-like plates can pass into the bone-like matter. I must adopt this opinion; analogy tends to confirm it. You will certainly see lymph deposited upon the serous membranes, occasionally converted, by a gradual change, into a bone-like substance. That most of these bone-like formations in the arteries have their origin in the atheromatous deposit, I have no doubt; but that lymph may occasionally assume the same appearance, appears to me equally certain.

In a great majority of these cases, you will notice the consequences of inflammation, rather than its actual existence. Occasionally, recently effused lymph is observed upon the interior surface of the artery, and also a degree of redness, not vascular, but diffused over the diseased portions. Sometimes, also, the edges of the excavations, formed by separated, or by separating bone-like masses, are swollen, and reddish in their hue. I believe it seldom happens that you can detect any distinct vascularity in the tissues affected. Indeed, it is well known that imbibition of blood, or even exposure of the opened vessel to the air, are the usual causes of redness of these tissues.

The causes of chronic inflammation of the aorta are very obscure. Among those which have been assigned, the abuse of ardent spirits is the most prominent. But in relation to this statement, I have no certain data.

The very exact and extensive observations of Bizot have proved that all portions of the thoracic aorta are not equally subject to the organic lesions that lay the foundation for aneu-

rism. Their most frequent seat is the posterior surface of the artery, which, when compared with the anterior surface, is found to be diseased in the proportion of 103 to 27—an extraordinary difference, and which would lead you to expect to find an aneurism springing from the posterior surface, much more frequently than from the anterior surface. Other portions most frequently affected, and I mention them in the order of their frequency, are the orifice of the coronary arteries, the portion of the aorta behind the sigmoid valves and the arch, especially the commencement of it. I would remark, however, that the difference in the frequency with which these different portions of the aorta are affected, is not very striking, certainly not sufficiently so to be of any great value in the diagnosis of the seat of an aneurism.*

If you review the different organic lesions that have been found to be associated with aneurism, and study the effects which they may produce in the physical condition of the aorta, you will easily perceive that the early stage of atheroma and a moderate degree of chronic inflammation, will operate chiefly in impairing the natural elasticity of the walls of the vessel, and that this may be general, for a certain distance in the course of the artery, or partial at a certain spot. You will understand how, in the first-named condition, the whole tube will be dilated, and, if equally so, the enlargement will assume a cylindric form, constituting the *cylindric aneurism* of Breschet, the *aneurism by dilatation* of other writers.

In cases in which the dilatation is partial, all the coats will be forced out from the side of the vessel into a sort of pouch, with a very wide mouth. This form has been called the true aneurism, which, indeed, is but a variety of that just mentioned. Some of the best pathologists have doubted its existence. It is stated by Scarpa, that the internal coat possesses no extensibility, and therefore cannot be pushed into a sac; while the middle coat, although capable of more distension, must yet soon give way as the sac enlarges. There can be no

* In 87 cases of aneurism of the thoracic aorta, 40 were of the ascending portion; 31 of the arch; 16 of the descending portion.

In 32 cases the heart was natural; in 30 cases it was enlarged; in 2 cases it was fatty.

doubt that there is much truth in this statement. It most cases, as Bizot remarks, in which the aneurismal sac is lined by a smooth and transparent false membrane, this has been supposed to be the inner arterial coat, while the existence of atheromatous spots under it has been regarded as evidence that the middle coat is there also. I have, I believe, seen one instance of a true aneurism of small size, of the ascending aorta, and supposed that I had seen others, before the fact that the sac might be lined by a transparent false membrane with atheromatous patches beneath it, was clearly made known to me.

The most common, as well as most formidable form, is that known as the *false* or *saculated aneurism*. In this form, the internal coat is entirely destroyed, and the middle coat partially so over a limited space varying from the size of a split pea to that of a dime, by the atheromatous degeneration; so that the column of blood, impinging upon this weakened spot, forces out what remains of the middle coat, as well as the external coat, into a sac, which often attains a great size, and communicates with the cavity of the vessel by a comparatively small orifice. This orifice, no doubt, enlarges as the column of blood presses upon it, so as to become as large as a quarter, or even half a dollar. It is usually rounded, with smooth, elevated edges formed by the middle coat, which here either ceases abruptly, or is continued growing more and more thin, to a certain distance into the cavity of the sac.

In other cases, this kind of aneurism originates in a different manner. The coats of the artery, from chronic inflammation, not only lose their elasticity, but become brittle, so that the column of blood impinging upon them, the internal and middle coats crack, and the blood forcing itself into the fissure, distends the external coat into a sac. At an early period, the elongated character of the opening would reveal the nature of the accident; but in more advanced cases, the opening becomes more and more rounded by the pressure of the blood, until at length it presents no unusual appearance. Occasionally, you may suspect, during life, that the aneurism has been formed in this way. Patients sometimes state that they have experienced a sudden "giving way," a sensation of rupture in the chest, after a muscu-

lar effort, or fit of anger, and that the symptoms of aneurism have soon followed.

The walls of the sac, when its growth is not very far advanced, is formed by the distended and thickened external cellular coat, often lined by a delicate and transparent false membrane, and near the orifice, sometimes, by what may remain of the middle coat. It may present numerous spots of atheroma beneath it, and patches of opake lymph upon its surface. But the external wall of the sac is no longer formed by the external coat of the artery when the aneurism is large. Condensed and thickened by the deposit of lymph in its areolæ, it is finally absorbed by continued pressure; but not before the surrounding tissues, also condensed and made adherent to it by a similar deposit of lymph, are ready to form a part of the sac. Thus, in its progress, portions of condensed lung, portions of the vertebræ, or of the ribs, which are, however, rarefied rather than condensed by the inflammatory action, the external muscles and integuments are made to enter into the structure of the sac. It is singular that while the bones are rarefied and rendered carious by the pressure of the sac, the intervertebral cartilages remain untouched, often standing out prominently, when the bodies of the vertebræ are nearly destroyed. The enormous size which these false aneurisms sometimes attain almost surpasses belief. I have seen one capable of containing two or more gallons of fluid.

But while the false aneurism tends to grow so rapidly, there is a compensating principle in it which should be carefully remembered, as it has a decided influence on the treatment; and this is the tendency to form coagula in the interior of the sac. It must be evident at once, that the influence of these coagula must be to diminish the pressure of the impinging blood, and this in proportion to their thickness and their elasticity. Indeed, there can be no doubt that the sac is occasionally filled up with these coagula, which become firm and organized, while the opening to the sac is closed by a delicate and transparent false membrane, leaving a slight depression to mark the spot where it existed. Corvisart saw tumors left by this curative process, and supposed them to be fibrous tumors attached to the aorta. Mr. Hodgson was the first to point out their true character. Nature

seldom, unfortunately, effects this desirable result, although in many cases you will notice a tendency in this direction. In some cases, you will find only a few coagula deposited in patches upon the interior of the sac; while in other cases, a firm coagulum lines nearly the whole sac, which when examined is found to be composed of more or less numerous concentric lamina, united to each other and to the sac by a loose kind of flocculent tissue. The older and most external of these layers are pale or rosy, and somewhat friable, while the internal, progressively, present the characters of recent coagula. It has been stated, that the more external of these layers sometimes present imperfect traces of organization. I have never seen this appearance; but it will not appear to you incredible when you remember that an aneurism may be transformed into an organized tumor.

The formation of these coagula evidently depends upon three causes: First, the stasis of the blood, which, entering the sac by a small orifice, and usually at a considerable angle with the arterial current, is compelled to stop there. The want of elasticity in the walls of the sac would favor the same result. Secondly, the inflammatory processes, which are constantly going on in and around the sac, tend to favor coagulation. And, thirdly, the presence of a good deal of fibrine in the blood. Thus in the aneurism by dilatation of all the coats, no clots are found; all of these conditions, except the last, being absent, or existing only in a trifling degree.

It is not uncommon to notice what has been properly called a *mixed aneurism*—a false aneurism engrafted upon an aneurism by dilatation. The progress of such an aneurism is necessarily slow and obscure at the commencement, until, by the destruction of the internal and the middle coats, all the formidable consequences of the false or saculated aneurism ensue.*

* In 82 cases of aneurism of the thoracic aorta, 64 were cases of false or saculated aneurism; 10 cases of mixed aneurism, cases which may be very well classed with false aneurism. Indeed, perhaps a large proportion of cases of the false aneurism might, in strict pathological language, be called mixed aneurism, the trunk of the artery from which the aneurism springs being usually more or less dilated. Finally, 8 were cases of simple dilatation. We find, then, in 82 cases, 74 cases of false aneurism, and only 8 cases of aneurism by dilatation of the aorta.

Finally, there is a form of aneurism of the aorta which may be regarded as a kind of pathological curiosity, since we know but little of its causes or of its symptoms. This form was first described by Mr. Skeleton of Dublin as the *dissecting aneurism*, and was so named by him from a supposed separation or dissection of the middle coat from the external coat. Judging from the two cases that I have examined, Mr. Skeleton's description is incorrect. In these cases a recent fissure was noticed near the commencement of the aorta, which had penetrated the internal coat, and, to a certain depth, the middle coat. Starting from this fissure, the middle coat was split, so as to form a canal filled by blood. In one of the cases this splitting of the middle coat was so considerable as to extend nearly to the diaphragm, and it embraced one-half the circumference of the artery. Indeed, in this case another fissure was noticed below, opening into the cavity of the vessel from the false passage, so that in fact the blood may have circulated through the whole length of the thoracic aorta in two parallel channels placed side by side. Whatever may be the cause of this singular change, it was evidently of recent formation. The lining membrane presented no unusual appearances, while the middle coat, although deeply stained by blood and evidently softened, still preserved its fibrous appearance. In these cases death was sudden, from the rupture being continued into the pericardium, which was found filled with blood, and in one case it occurred after a considerable muscular effort.*

* In an analysis of 89 cases of aneurism of the thoracic aorta, I find six cases of the dissecting aneurism. In four cases the patients were males, in two cases females. Their average age was 56 years. Five commenced in the ascending aorta, extending in one case to the origin of the renal arteries. In one case the descending aorta was the seat of the disease, which ruptured the left pleura. The five cases which commenced in the ascending aorta all ruptured the pericardium.

There is in the museum of this Hospital a small aneurism of the coronary artery which had ruptured into the pericardium. I have seen also a small aneurism of the mitral valve. A patient with cerebral disease, age not mentioned, was admitted into this Hospital under the care of Dr. Post. No disease of the heart was suspected. The heart was enlarged, especially the left ventricle. The aortic valves were covered by soft vegetations. An aneurism of the mitral valve, the size of a small cherry, projected upward into the left auricle. It contained a clot

The changes which exist in the arteries, and which, taken together, grow more frequent as life advances, are about equally common in males and in females. You might readily infer from this that aneurism would be found in about equal proportions in the two sexes. But this is far from being the fact. In ninety-four cases of aneurism of the thoracic aorta analyzed by myself, and in which a post-mortem examination was made, eighty occurred in males, and only fourteen in females.*

How, then, will you explain the great predominance of aneurism in the male sex? It has been supposed that the greater exposure of this sex to violent efforts, by which the internal and middle coats of the diseased artery might be ruptured, might thus form the commencement of an aneurism. This I believe to be true; but will it explain the striking difference which I have noticed? A violent effort would most probably produce a false aneurism. In thirteen, however, of the fourteen cases of aneurism of the thoracic aorta in females which I have analyzed—in a single case the post-mortem examination was unsatisfactory on the point in question—false aneurism existed in every instance. A violent effort, indeed, is in no way essential to the production of a false aneurism. When a limited surface of the arterial coats is destroyed by atheromatous softening, the ordinary impulse of the blood is quite enough to cause an aneurism.

But the organic changes which lead to the formation of aneurism become more abundant and more frequent with the progress of age. You would, then, expect to find aneurism rather a disease of advanced life. But this pathological fact, like that relative to sex, is not entirely confirmed by experience. Thus,

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of coagulated fibrine. It communicated with the left ventricle by a considerable aperture, and at its upper portion it opened also into the left auricle by a small fissure.

* Mr. Hodgson's experience quite agrees with this statement. He had seen 63 cases of aneurism, 56 of which existed in males, and only seven in females. Twenty-nine of these 63 cases were aneurism of the thoracic aorta, and six of the seven cases which occurred in females were seated in the same vessel.

The Report of the Inspector of the City of New York, during three successive years, gives only 27 cases of aneurism. Of these, 20 occurred in the male sex.

The statistics of the Registrar for London gives a different general result. Thus of 100,000 persons born in London, 61 die of aneurism; 29 males, 32 females.

in eighty-five cases of aneurism of the thoracic aorta analyzed by myself, the average age was but forty-one years.* Thus, aneurism of the aorta is a disease of middle life, and while the system is still in full vigor. This want of correspondence, then, between the organic lesions which produce aneurism and the actual existence of the disease, is noticed in relation to age, as well as in relation to sex, but not so strikingly. Again, you will see in this view, the same probable influence which I alluded to before—the influence of violent efforts in the production of aneurism. Thus, although the organic changes which favor the production of aneurism are more marked in those advanced in life, yet violent and frequently repeated physical effort is much less frequent. Taking, then, the two facts that I have mentioned,—viz., that females, while they are equally disposed with males to the arterial changes which induce aneurism, yet seldom suffer from the disease—and that, again, while these arterial changes are decidedly most marked in advanced life, yet aneurism is rather a disease of middle life,—it does not appear to me unreasonable to suppose that active physical efforts, so much more frequent in the male sex during the vigorous period of life, are a powerful cause in the production of aneurism.

* According to the Registrar-General, of 100,000 persons born in London, 61 die of aneurism: 4 under 20 years of age; 16 between 20 and 40 years; 41 after 40 years. This statement gives a greater influence to advanced age than that derived from the cases I have analyzed. But the latter have this advantage—they were all confirmed by post-mortem examination.

LECTURE XXXV.

ANEURISM OF THE THORACIC AORTA.

The false, or saculated aneurism.—Symptoms: physical signs, progress and termination of the disease.—Aneurism by dilatation, or true aneurism.—Symptoms and physical signs.—Aneurism of the pulmonary artery.—Aneurism of the arteria innominata.—Constriction of the aorta.—Treatment of aneurism.

In calling your attention to the symptoms and the progress of aneurism of the thoracic aorta, I shall first speak of that form which is not only the most formidable and frequent, but most distinctly marked during life—the false or saculated aneurism. Indeed, I may state as a general fact, that the other forms of the disease seldom reveal themselves by distinct symptoms during life. They usually attain to so small a size, and are surrounded by organs so yielding in their structure, that symptoms of slight pressure, of obscure and ill-defined oppression in the chest, is all that attracts attention. The careful auscultation of the chest will sometimes reveal the true nature of the disease, when it is resorted to; but this is frequently of no avail. Not so with the false aneurism. Its rapid growth, the destruction that accompanies its progress, the severe pain that attends it, and, finally, the existence of an external tumor rapidly increasing in size, will often lead you directly to a careful study of the cause of such marked symptoms, and to the more frequent detection of the disease.

When a false aneurism has attained only a moderate size, its symptoms are those of obstruction and of pressure; indicated by dyspnœa, when the trachea or larger bronchi are compressed, by lividity and œdema of the face, when the vena cava suffers from the same cause; or by dysphagia, when the œsophagus is obstructed. These symptoms, although likely to increase by the progress of the tumor, are by no means equally marked at all times, or even constant in their existence. The intermittent character of the symptoms induced by the pressure of all organic tu-

mors is worthy of remark. In the case of aneurism, they vary with the position of the patient, with the force of the circulation by which the pressure may be increased or diminished; they vary also from causes not always easily explained. But in an aneurism, it is easy to understand that the pressure will vary with the quantity of blood the sac may contain, and that this will vary, and often rapidly, with the varying force of the action of the heart.

In some cases, the pressure on the trachea leads to hoarseness, loss of voice, a hoarse sonorous cough, often with frothy mucous expectoration, and sometimes to symptoms of sudden suffocation; so that tracheotomy has been performed in the vain hope of relief. I have related a remarkable case of this kind, in my lecture on laryngitis. It is probable that the affection of the voice in these cases, may be sometimes owing to pressure on the recurrent nerve, thus inducing a paralysis of the muscles which act upon the vocal chords. Pain, induced by the disorganizing influence of the tumor, and still more, by irritation of the spinal nerves, is often an early and severe symptom of the false aneurism. These pains are of a neuralgic character, shooting from the spine around the chest, and sometimes resembling those noticed in angina pectoris. No doubt also the nerves of organic life, the branches of the sympathetic nerve, are often irritated, and thus add to the distress of the patient. It has been doubted whether these last-mentioned nerves are capable of inducing the sensation of pain. They probably are not, except through the branches of the spinal nerves with which they are connected. To these symptoms may be added a sense of internal pulsation, remote from the heart.

If now you search for physical signs of disease, and the tumor be still of moderate size and deep seated, you may find but little aid to the diagnosis from this source. Careful palpation may discover a pulsation at a distance from the heart; there may be slight dulness on percussion over a limited space, and on applying the stethoscope, you may hear a blowing sound, accompanying the first sound of the heart, having its maximum, not over the valves of the heart, but more or less remote from them; or you may hear nothing of the kind. When, however, the tu-

mor by its growth becomes more superficial, these physical signs not only become more constant, but more distinct, and new signs are added, which may change a doubtful case into an absolute certainty. An external tumor may gradually present itself; pulsating, expanding to the touch, while the dulness on percussion and the blowing sound become still more evident. The usual seat of these external tumors is, for aneurism of the ascending aorta under the second and third ribs to the right of the sternum, and for the arch under the upper portion of the sternum. Sometimes an aneurism of the left portion of the arch will present to the left of the sternum, also under the second and third ribs, while aneurisms of the descending aorta usually present themselves externally, near the spine, or under the scapula. The precise direction that an aneurismal tumor will take could hardly be estimated, even if you knew precisely its origin. You know this, however, as the result of general observation, that the organic changes leading to the formation of aneurism are far more frequently situated upon the posterior portion of the aorta than anywhere else. Thus it is fair to infer, in a majority of cases, that the early progress of the tumor is backward, in the direction in which the trachea is most likely to suffer from pressure, in which also the nerves are most likely to be irritated, and in which, finally, an external tumor is the least likely to appear. The history of individual cases, in which disturbance of the respiratory function and pain are the earliest symptoms, and an external tumor among the last that develop themselves, may thus be easily explained. Indeed, in a majority of cases no external tumor is discovered.*

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* In about 90 cases of aneurism of the thoracic aorta which I have examined, an external tumor is mentioned 18 times, viz.: in 10 cases of aneurism of the ascending portion, in 4 cases of aneurism of the arch, in 3 cases of aneurism of both the ascending portion and the arch, and in one case of aneurism of the descending portion.

The following statement will illustrate more fully the seat of the tumor in these cases, and its connection with the artery:

Case 1st. Ascending aorta, upper portion: middle one-third of sternum, and cartilages of the 2d and 3d ribs on both sides.

Case 2d. Ascending aorta, upper portion: to the right of the sternum, between the 2d and 3d right sterno-costal articulations.

The physical signs of an external circumscribed tumor, pulsating with the heart and expanding to the touch, accompanied by a thrill, by dulness on percussion, and by a blowing sound synchronous with the impulse, preceded and accompanied by the symptoms of internal pressure, will sometimes render your diagnosis of false aneurism of the thoracic aorta easy and certain. If the tumor in its progress has dislocated the clavicle, or the ribs, you have a new fact added of much importance; yet none of these physical or rational signs are constant, and with, perhaps, the exception of that last mentioned, they may exist independently of an aneurism.

A pulsating tumor, elastic and expansive to the touch, has been justly regarded as a strong indication of an aneurism.

Case 3d. Ascending aorta: at the junction of the 3d and 4th left ribs with the sternum.

Case 4th. Ascending aorta: to the right of the sternum, over the 2d or 3d costal cartilage.

Case 5th. Ascending aorta, upper portion: under right clavicle.

Case 6th. Ascending aorta; upper half of the right side of the chest in front: pointing at the middle of the sternum, close to the median line.

Case 7th. Ascending aorta, upper portion: under the cartilages of the 2d and 3d left ribs.

Case 8th. Ascending aorta: an inch to the right of the sternum, in a line with the nipple.

Case 9th. Ascending aorta: under the upper one-third of the sternum, inclining to the right side.

Case 10th. Ascending aorta: same situation as in case 9th.

Case 11th. Arch: behind the sternum and on the left side, from the upper edge of the cartilage of the 2d rib to the corresponding portion of the 1st rib, and in the left supra-clavicular space.

Case 12th. Arch: behind the centre of the sternum.

Case 13th. Arch: behind upper part of sternum, inclined to the right side (the innominata was also aneurismal).

Case 14th. Arch: top of sternum.

Case 15th. Ascending portion and arch: upper portion of sternum.

Case 16th. Ascending portion and arch: top of the sternum, extending to each side, and especially to the right side.

Case 17th. Ascending portion and arch: between the 2d and 3d left costal cartilages.

Case 18th. Descending aorta, upper portion: under the cartilages of the 1st and 2d left ribs, close to the sternum.

In another case of aneurism of the ascending aorta alone, the tumor also presented on the left side, under the cartilages of the 2d and 3d ribs.

But if you allowed yourselves to be guided by these signs alone, you might be mistaken. I have seen an abscess produced by caries of the sternum and situated near the heart, produce precisely such a tumor. Cases are recorded in which the pointing of an empyema has also produced such a tumor. Indeed, I believe that a solid tumor, if situated over a large artery, may present the same indications, even to the expansive feeling. A blowing sound, heard at a distance from the heart, has often led to the same conclusion that aneurism existed. Yet this blowing sound may not be heard, and still an aneurism may exist; or the reverse may happen, the sound may be heard, and yet no aneurism be found. It is important to examine the causes that produce and modify this sound before you can understand its true value in the diagnosis.

The blowing sound may exist as a simple blowing sound, or as a sawing or rasping sound, but usually its tone is somewhat peculiar, and may be called whizzing. It is produced by the disturbance in the current of the blood as it enters the orifice of the sac. Its primary seat is at the orifice, although new vibrations may be generated by the rush of the blood over the rough walls of the sac, and these may be increased in loudness by resounding in the cavity of the sac. If the orifice of the sac be small you may hear no sound, for the sac may thus be kept constantly so full of blood, that enough of this fluid cannot enter with each contraction of the heart to generate a sound. Again, the same result may happen in a different way. If the orifice of the sac be very large, the current may not be sufficiently disturbed to generate much vibration, and what is produced is diffused over so large a surface as not to reach the ear. But this is not all. The condition of the sac itself influences the sound. Thus, if the sac be nearly full of coagula, so little blood will enter the orifice that no sound will be generated, and if these coagula are soft and inelastic, a feeble sound would be lost before it could reach the ear. Finally, the force of the impulse of the heart must be considered; for if the action of the heart is feeble, however favorable other circumstances may be, no sound may be generated. The same reasoning will apply also to the thrill felt by the touch. The same vibrations which

generate the sound generate also the thrill, but only the more intense and more readily transmitted vibrations. So that you may have a blowing sound and no thrill, but never the reverse.

This blowing sound, so far as my knowledge extends, is frequently absent in thoracic aneurisms—but when it occurs I have always found it single, accompanying the impulse of the heart. Yet it has been described as being sometimes double; and lately I have read cases in which it has been described as occurring only with the second sound, or with the diastolic sound of the heart. I must confess that I am incredulous in relation to both these points, and especially to the last. I can, indeed, conceive it possible, that the elasticity of the sac may be such as to expel a portion of the blood during the diastole of the heart, with sufficient force to generate a murmur, but such cases must be rare. But how shall we account for the sound with the diastole alone? It has been stated that in these cases the orifice of the sac is large, so that no sound is generated when the blood enters it, but that what remains of the coats of the artery, just within the orifice, is sometimes thrown up so as to form a kind of valve, acting in the direction from the sac towards the artery, so that the orifice is narrowed during the egress of the blood, and thus a diastolic sound is generated. But this opinion requires revision. In a very large proportion of cases, the sound is single and sytolic—that is, it accompanies the systole of the ventricle and the pulsation of the tumor.

I have stated that this single systolic sound, and having its maximum remote from the valves, may occur without an aneurism. A contraction of the aorta at a certain point will produce it. Any tumor, as an enlarged gland, by compressing the artery at a certain point, will lead to the same result. If the tumor presents itself externally, the difficulty in the diagnosis may still be considerable. Finally, the blowing sound may exist, and even be accompanied by a thrill, without any organic disease at all, as in cases of chlorosis and of anemia. In the latter case, however, there is no tumor, and the sound usually commences at the origin of the aorta, and extends upward in its course, instead of being limited in extent, and more or less remote from the heart, as in the case of an aneurism.

An aneurismal tumor produces upon the neighboring organs the symptoms of pressure, some of which deserve a more particular notice. The vena cava descendens, lying by the side of the ascending aorta, and the left vena innominata, lying just above the arch, may readily become compressed by the tumor, or even more or less completely obliterated by a clot in their cavity. As the result, the patient may have headache, lividity of the face, œdema, and even apoplexy. Compression of the trachea induces a peculiar whistling respiration, or wheezing, like that of asthma—symptoms of strangulation, hoarseness, and even aphonia, which has been attributed to a paralysis of the muscles of the larynx supplied by the recurrent nerves. These symptoms have not unfrequently been misinterpreted by being referred, as I have already remarked, to disease of the larynx itself. In urgent cases, the operation for tracheotomy has been performed, and of course, without benefit. These symptoms, although the result of pressure, are often intermittent, and remarkably relieved or aggravated by position, according as the pressure is thus increased or diminished. The auscultation of the lungs will sometimes afford you important aid in the diagnosis. When the trachea is compressed, the respiration may be feeble over the whole chest. Where a single large bronchus is affected, a limited feebleness of the respiratory murmur has been noticed, with a puerile respiration over the other portions of the chest. Usually the percussion sound is natural over the whole chest, except at the spot where the aneurismal tumor reaches the surface of the chest, and where there is more or less dulness over a limited space.

Among the symptoms occasionally produced by the pressure of an aneurism is an inequality of the pulse, according as the right or the left subclavian artery is compressed. I have met with a case in which both these arteries were affected, so that no pulse existed at either wrist. The pulse in the right wrist is probably most frequently affected.

A sailor, about forty-five years of age, entered this Hospital, after complaining for some months of cough, wheezing respiration, and of occasional paroxysms of dyspnœa, especially at night, which usually subsided with a free expectoration. The chest was everywhere resonant on percussion, and a sibilant or

a sonorous rhonchus was universal. No physical signs of an aneurism were detected, but it was noticed that the pulse was absent in both wrists. The patient, after remaining a few weeks in the Hospital, died suddenly.

A false aneurism of the arch of the aorta, capable of containing a small orange, existed. It contained a thin stratum of old coagula, and communicated with the artery by a large opening, the size of half a dollar. The arteria innominata was obliterated at its origin. The left subclavian artery was compressed by the tumor. At the commencement of the descending aorta another false aneurism existed, also communicating with the artery by a large opening. When the chest was opened, a singular appearance presented itself. The cavity of the right pleura was filled by a dark-red mass, looking very much like a mass of currant jelly, and covered by a thin and transparent membrane. This was formed by the right pleura costalis having beneath it an immense recent coagulum of blood. The inferior aneurism had opened into the posterior mediastinum, and had separated the costal pleura to a great extent. The lungs were very pale, and were somewhat emphysematous. The bronchi were inflamed. The left ventricle of the heart was hypertrophied.

It has also been noticed that death from inanition may ensue from the pressure of an aneurism on the thoracic duct, or upon the œsophagus.

An aneurism, by disorganizing the lungs, may induce a distressing cough, with a muco-purulent expectoration. I have known a patient who expectorated suddenly a large quantity of this mucus, with an apparent diminution of the tumor; the matter being mistaken for pus. The tumor was supposed to be an abscess. The disorganizing process, however, does not tend to suppuration, but to adhesion and to ulceration. The absence of pus, indeed, has led some pathologists to regard the destruction of the bones by aneurisms, not as the result of inflammation, but of mechanical attrition.

A case of saculated or false aneurism of the ascending aorta, once presented itself to me, in which the diagnosis was attended with considerable difficulty. There were some striking reasons for supposing that the case might be an abscess.

A gentleman, about thirty-five years of age, of good constitution, and while in the enjoyment of good health, became suddenly affected with a feeling of painful constriction passing through the chest from the cartilage of the second to that of the fourth rib, back to the scapulæ, and principally on the right side. The pain also extended to the right shoulder. This pain soon subsided, and for two months the patient thought himself quite well. But the pain returned in the right shoulder, extending up the neck and down the right arm. It was of a sharp, shooting character, and was exceedingly distressing. After a time a dry cough supervened; the neuralgic pains continued, but with decided remissions. About six months after his attack he began to notice a pulsation in the upper portion of the right side of the chest, and to experience dyspnœa with exercise. His breathing at times was asthmatic. Nine months after his first attack, a tumor about the size and the shape of a walnut, and pulsating strongly, was noticed in the right omo-clavicular space, and the skin covering it became red and tender. In the course of ten days this tumor moved, gradually, from its first location, across the neck to the hollow above the sternum, where it pulsated strongly, and appeared of the size of the largest extremity of a hen's egg. The neuralgic pain continued severe, the dyspnœa was decided, and the cough was frequent, loud, dry, and often long continued and distressing. After a considerable interval, an abundant purulent, offensive expectoration more or less tinged by blood, suddenly occurred. It varied in quantity from a pint to a quart in a day. With this expectoration, the neuralgic pain abated, the dyspnœa diminished, the tumor decreased in size, and the pulsation was less marked. The appetite and the strength also improved, so that the patient thought himself nearly well. But after a few weeks, the pain increased again, the pulsation and the tumor also increased, and the right arm, for the first time, became œdematous. Again the copious expectoration of purulent matter occurred, but without much relief.

It was at this time that I first saw the patient, exactly a year after his first attack. His aspect was pale, but bright and cheerful; he had emaciated somewhat. He had no hectic symp-

tions, his pulse was feeble, 108 in a minute, and equal in both wrists and temples. The action of the heart was regular, but feeble. On exposing the chest, an egg-shaped tumor, with its larger extremity towards the right shoulder, seven inches in length by three inches in breadth, extended from the middle of the clavicle on the right side to the inner edge of the middle of the sterno-cleido-mastoid muscle on the left side. The right clavicle, dislocated at its sternal extremity, and elevated, passed over the middle of the tumor like a bridge. The upper bone of the sternum was also pushed forward, but in a less degree. The right sterno-cleido-mastoid muscle passed over the tumor and was lost in it, and a line of pulsation along the course of the right common carotid artery was also noticed. The trachea was pushed to the left side. The tumor was extremely elastic to the touch, it pulsated strongly in every part, and especially immediately over the sternum. Its right portion was more firm, and pulsated less than other portions of the tumor. There was no bellows murmur or thrill. The surface of the tumor presented a faint blush, but without heat or marked tenderness. It was slightly œdematous. The whole superior portion of the right side of the chest was dilated anteriorly; this dilatation ceased abruptly at the level of the second rib. This side of the chest was also œdematous. The movement of the shoulder-joint was limited, from a feeling of stiffness.

The percussion over the chest was natural; but the respiration was bronchial at the summit of both lungs, especially of the right lung, while it was wheezing and rough in the other portions of the chest, but without mucous rattle. The voice was hoarse, the respiration wheezing, the cough frequent and croupal, and attended by a considerable muco-purulent expectoration partly tinged by blood. The cough was relieved by talking and by mental diversion. Emollient warm applications to the tumor gave decided relief to the pain.

Several distinguished practitioners saw this case in consultation, and doubts were entertained whether the tumor was an aneurism or an abscess.

The facts in favor of an aneurism were, the long duration of the disease, the absence of hectic symptoms, the dislocation of

the clavicle, the pulsation of the tumor. The facts in favor of an abscess were, the remarkable change in the position of the tumor, the decided subsidence of the tumor and the diminution of the pulsation with a relief of the neuralgic symptoms, all coinciding with a sudden expectoration of what was said to be purulent matter, but which was probably only muco-purulent matter.

The patient dying a few days afterwards, the tumor was found to be a false aneurism of the ascending aorta, communicating with the vessel by an opening about the size of a quarter of a dollar, and with smooth and even edges. The tumor originated from the artery, just below the arteria innominata, so that this vessel and the right subclavian artery opened into the sac. The cavity of the tumor was half filled with soft coagula. The right clavicle and the upper bone of the sternum were carious, and adhered to the walls of the sac. The heart was healthy. The lungs were generally adherent by old adhesions. They were not disorganized or much compressed, but were congested with a bloody, frothy serum. There were the evidences of hepatization in the lower lobes. The bronchi were dilated and somewhat inflamed.

The false aneurism tends to rupture and to death by hemorrhage. The tumor, when it exists externally, may assume over its most prominent portion a reddish tint, which soon becomes purple, then a small slough may form and hemorrhage ensue. But this is a very rare accident. In eighty-seven cases of aneurism, in which there were fifty-five ruptures, only one was ruptured externally. I have noticed in these cases of aneurism forming an external tumor, that death ensued from exhaustion, or from internal hemorrhage, at about the time that an external rupture might have been looked for. Very frequently indeed, however, the aneurism bursts internally—fifty-four times in eighty-seven cases—having encountered in its progress some of the hollow organs in the chest. Aneurisms of the ascending aorta burst most frequently into the pericardium; those of the arch into the trachea; and those of the descending thoracic aorta into the left pleura. Hæmoptysis, or hæmatemesis, may follow, which may be sometimes moderate in degree, and repeated until death

may ensue gradually. Or it may occur suddenly, with profuse hemorrhage, with symptoms of suffocation or of collapse with internal hemorrhage.

A singular case occurred in this Hospital, several years ago, in which the rupture of the aneurism took place in the spinal canal, inducing sudden paraplegia. Of all these modes of death, none are more interesting than the rupture of an aneurism of the ascending aorta into the pericardium. Many cases of sudden death are owing to this cause, and often without the disease being clearly indicated during life. Indeed, the aneurism is usually small, owing to the pericardium supplying the place of the external coat of the artery at its origin, and which is easily ruptured by pressure.*

A seaman, aged thirty years, was brought to this Hospital. He complained of pain in the epigastrium, and of great dyspnœa. About three hours after his admission he died suddenly.

On examining the chest, the pericardium was found distended with about two pints of blood, partially coagulated. An aneurism of the aorta, the size of a horse-chestnut, existed at the origin of the vessel. It communicated with the pericardial sac by an opening large enough to admit a probe.

There is in the museum of the Hospital a specimen, which, so far as my knowledge extends, is unique. An aneurism of one of the coronary arteries, about the size of a pigeon's egg, had ruptured into the pericardial sac. The case was that of a young

* In 40 cases of aneurism of the ascending aorta, 19 were ruptured; 16 were of the false variety; one an aneurism by dilatation; and in two cases their character was doubtful. Of these—7 ruptured the pericardium; 2 the right ventricle; 2 the right auricle; 2 the vena cava superior; 1 the pulmonary artery; 1 the œsophagus; 2 the right pleura; 1 the left pleura; 1 the right lung.

In 31 cases of aneurism of the arch, 20 were ruptured. Of these—16 were cases of the false aneurism; 3 of the aneurism by dilatation; in one case the character was doubtful. Of these—6 ruptured the trachea; 3 the œsophagus; 3 the left pleura; 3 the pericardium; 1 the left bronchus; 1 the left lung; 1 the pulmonary artery; 1 the posterior mediastinum; 1 externally.

In 16 cases of aneurism of the descending thoracic aorta, there were 20 ruptures: 15 were the false aneurism; 1 of the aneurism by dilatation. Of these—5 were ruptures of the left pleura; 3 of the left bronchus; 3 of the œsophagus; 2 of the left lung; 2 of the trachea; 2 of the right lung; 2 of the posterior mediastinum 1 of the pericardium.

lady, who had just been dressing herself for a ball. In descending the stairs from her chamber, she died suddenly.

Such is the progress and the termination of that most formidable variety of aneurism, the saculated, or false aneurism. In the *aneurism of dilatation* of all the coats of the vessel, you have a much less serious disease to encounter. It may justly excite your fears, if you have recognized the disease, that a false, or saculated aneurism, may become engrafted upon it, but while it remains as a simple dilatation, it usually produces but few symptoms, and these are by no means of a striking character. Hardly ever attaining to more than two or three times the natural size of the artery, and surrounded by organs disposed to yield to its uniform and diffused pressure, it may induce more or less oppression in the chest, but often nothing more. Sometimes it presents itself as an external tumor above the clavicles, or the top of the sternum, appearing rather as a fulness than as a distinct tumor, with the characteristic, expansive pulsation, and often with a thrill. It is in these cases, that the modified blowing sound shows itself in its most marked form. The column of blood dashing over the internal coat of the dilated vessel rough with atheroma, with bone-like or cartilage-like deposits, and without coagula to break the sound, produces a loud, hoarse, resounding murmur, far more striking than that usually heard over the valves of the heart, or in the false aneurism. It is commonly single, and synchronous with the contraction of the ventricles. It may be double, the second murmur being produced, as the column of blood falls back upon the heart during its diastole. In cases in which the dilatation exists in the ascending aorta, or at the arch, as perhaps, also, in the false aneurism, you may hear distinctly the second sound of the heart, either pure, or modified by disease of the aortic valves.

If, then, you have a patient who has experienced, for a considerable time, dyspnœa, occurring, perhaps, in paroxysms, with a wheezing respiration, and accompanied by a hoarse, laryngeal cough, dry, or attended, perhaps, by a frothy mucous expectoration; if the voice is sometimes hoarse or feeble; if the patient complains of dysphagia; if the veins of the neck, and especially on one side only of the neck, are turgid, perhaps with

œdema of the parts, or of the corresponding side of the face and the arm; if there is severe neuralgic pain in the chest, especially on one side, perhaps with a corresponding tender point over the spine; if the pulse at one wrist, especially at the right wrist, is more feeble; if the resonance over the chest is natural, while the respiration is feeble over a limited portion of one lung, perhaps over the superior lobe, or even over the whole lung, and sometimes accompanied by a loud, but limited, sonorous rhonchus, while it is puerile in the other portions of the lungs—you may suspect that a tumor of some kind is compressing the organs in the chest. If, in addition, you can discover a deep-seated pulsation in the chest, more or less remote from the heart; if, with this, there is a blowing sound, also remote from the heart; if, again, over a limited spot, and especially over the anterior and superior portion of the chest, there is a limited dulness on percussion, and perhaps a thrill communicated to the touch; if, finally, an external tumor presents itself, especially in the same region—pulsating, expansive, increasing with considerable rapidity—then the evidences of an aneurismal tumor gain with the progress of the case a progressive certainty—a certainty which will be proportionate to the completeness and regular development of the indications I have pointed out. But you must remember, on the other hand, that the growth of an aneurismal tumor is capricious; that being surrounded by organs, more or less capable of displacement without alteration in their functions, and increasing sometimes more in one direction than in other directions, the symptoms of compression will vary very much from these causes; that as the tendency of aneurismal tumors of the thoracic aorta is to grow from the posterior wall of the vessel, there is a tendency to penetrate into the chest rather than to seek the external surface. Hence, they are often deep seated, and in a large proportion of cases, probably, do not present any external indications of their development, at least until a late period; and finally, that the seat of the tumor, in reference to the portion of the artery affected, modifies the symptoms in a remarkable manner. Thus an aneurism of the ascending aorta, or of the arch, is attended usually by more distinctly marked symptoms of compression, and by the physi-

cal evidences of the disease, than an aneurism of the descending thoracic aorta, removed from the trachea, and the œsophagus, and the great veins of the neck. But this fact, also, must be remembered: that aneurismal tumors in this lower portion of the aorta, and springing frequently from its posterior wall, are brought directly in contact with the spinal column; and that neuralgia and spinal tenderness are more likely to be early and more constant symptoms in an aneurism thus seated, than in the more common cases affecting the great vessel nearer its origin.

Aneurism of the pulmonary artery sometimes exists, but it is a very rare form of disease. I have never met with a case. In the 25th volume of the Medico-Chirurgical Transactions, a case is recorded by Mr. Fletcher, of dilatation of this artery with thinning of the coats. Malformation of the heart, valvular disease, as well as a stricture of the aorta, also existed, so that it is impossible to know exactly how much to attribute to the aneurismal dilatation. The constitutional and the rational symptoms were those met with in ordinary disease of the heart. Examining the chest, a very superficial pulsation with purring tremor, was felt between the second and the third ribs to the left of the sternum, and a loud, rasping, bellows sound was heard close under the stethoscope. This sound was heard all over the chest, except on the right side posteriorly, and it ceased abruptly above the clavicles. It is probable that these physical signs were dependent upon the aneurism of the pulmonary artery, especially as they agree with those noticed in the much more simple case recorded by Hope.

A female, 36 years of age, of a sallow complexion, complained of pain in the epigastrium and of dyspnœa. The catamenia had been suppressed for five months, at which time œdema of the feet occurred. She had suffered from dyspnœa for two years, caused by striking the breast against a post. The pulse was 70 in a minute, and large, full, and rather tense. The tongue was clean, the urine was scanty; ascites existed.

There was extended dulness over the precordial region, with a pulsation, thrill, and prominence between the cartilages of the

second and the third ribs on the left side. The impulse of the heart was much increased and extended, particularly in the left precordial region. It existed also in the epigastrium. The systolic sound was very loud, harsh, superficial, and sawing. Its maximum was at the prominence between the second and the third ribs, but it was much diffused. The patient lived a month.

By post-mortem examination, the heart was found generally enlarged, especially the left side. The pulmonary artery was remarkably dilated. Its internal circumference near the valves was four and a half inches, and midway between this point and its bifurcation, it was five inches. The dilatation did not extend beyond the bifurcation. The sigmoid valves of the pulmonary artery appeared insufficient to close the pulmonary orifice. The aorta was rather contracted, the mitral valve was slightly thickened. The peritoneal cavity contained three or four quarts of serum. The liver was rather enlarged and hard, and the peritoneum covering it was thickened by old inflammation.

In this case the blowing, or rather the sawing sound, was undoubtedly connected with the aneurism. Had it been dependent upon regurgitation through the pulmonary orifice, it would have been diastolic instead of being systolic.*

I have met with a case of partial dilatation of the aorta, also, just above the origin of the vessel, which I will mention, that you may compare it and contrast it with the physical signs exhibited in the case which I have just related. The physical signs of aneurism were remarkably alike in the two cases, except in relation to their seat.

The patient was a young sailor, who entered this Hospital with the physical signs of enlargement of the heart, and of imperfect closure in the aortic valves, the regurgitation being indicated by a diastolic filing sound. But, in addition, there was a distinct circumscribed pulsation to the right of the sternum, one and a half inches beyond the median line, and between the cartilages of the third and fourth ribs. Over this spot a loud, sys-

* A case is related in the London Lancet, in which an aneurism the size of a nutmeg, springing from a branch of the pulmonary artery the size of a crow-quill, opened into a tuberculous cavity at the summit of the left lung.

tolic, bellows murmur existed, and besides, a feeble and distant diastolic sound, which seemed to be transmitted from that heard over the aortic valves. The sound on percussion, over this spot, was dull.

By post-mortem examination, the evidences of enlargement of the heart, and of imperfection in the aortic valves, were quite apparent. The valves of the aorta were thickened, hard, and irregular in shape, and their ventricular aspect was covered by vegetations, which extended upon the lining membrane of the left ventricle. The valve most covered by vegetations was much shrunken and contracted. The aorta, two inches above its origin, was formed into a pouch capable of containing half a hen's egg, and which occupied the right lateral half of the vessel. This pouch was formed by a dilatation of all the coats of the vessel, which were generally thinned and dilatable. Its communication with the artery was free, the diameter of the point of communication being equal to the diameter of the pouch itself. There were a few atheromatous spots in the aorta.

Aneurism of the *arteria innominata*, a comparatively rare disease, may present itself as a pulsation and a thrill, united with a blowing sound and a dulness on percussion in the neighborhood of the right sterno-clavicular articulation. In time, the tumor may present itself externally above the superior and right edge of the sternum, extending up the neck along the inner edge of the sterno-cleido-mastoid muscle. This aneurism is frequently associated with a similar condition of the aorta in its neighborhood, either of the ascending portion or of the arch, and the coats of the vessels are usually much diseased, especially by atheroma. The bold and brilliant operation of Dr. Mott, for the cure of this aneurism by a ligature placed below its origin, is liable to failure, not merely from the nearness of the artery to the heart and its large size, but also from the very frequent disease of the coats of the vessel and of the aorta itself. Indeed, this operation, after several unsuccessful attempts to cure the disease, would hardly now be recommended by the judicious surgeon. There is, however, a successful case in which a ligature was placed upon the carotid and upon the right subclavian

artery: thus far successful, at least, that when the patient died of some remote disease, about a year, I think, after the operation had been performed, the aneurismal sac, much diminished in size, was found completely filled with a mass of fibrine, and was thus in progress of cure. But I am passing out of my province into that of surgery.

The opposite condition to an aneurism has sometimes been found in the thoracic aorta: *a stricture of the aorta.* This form of disease, which may be regarded as a pathological curiosity, is usually seated at the superior portion of the descending aorta. It may be connected with atheroma or other organic changes in the artery, but sometimes is unattended by any apparent disease of the coats of the vessel. The artery appears as if a ligature had compressed it. In Mr. Nixon's case, reported in the Dublin Medical Journal, the stricture appeared as if the edge of a knife had been pressed inward upon the external surface of the vessel, so as to diminish its calibre one-half. The coats of the vessel at the seat of the stricture presented no evidences of disease, although a few spots of atheroma existed in its course. The heart was enlarged, and the aortic orifice obstructed by vegetations and calcareous deposits. In a case reported by Dr. Clark to the Medical and Surgical Society of this city, many years ago, "the aorta showed no marks of disease; except that three and a half inches below the arch on the posterior and right side of the vessel, there was observed a cord stretching across a small portion of its calibre. This bridge was formed by a few fibres of the elastic coat, and was covered by the lining membrane of the vessel. It strikingly resembled in length and position one of the chordæ vocales of the larynx, and had under and behind it a little sinus, like the semilunar fossa."

The sign of this condition, in Dr. Clark's case, was a loud sawing sound, extending downward as far as the femoral arteries; while upward, at the arch of the aorta and over the heart, it was not heard at all. In Mr. Nixon's case, a loud bellows sound existed through the whole of the aorta, and there was a strong pulsation in the arteries of the neck; but as the heart was enlarged, and the aortic orifice obstructed, the bellows sound in

the ascending portion of the aorta and at the arch may have been produced by these causes.

It is remarkable, that, in both these cases, an abdominal aneurism was supposed to exist, and that in neither case was the fact verified by post-mortem examination. In Mr. Nixon's case, a pulsating tumor appeared below the margin of the right false ribs, and attained a considerable size; it had the purring thrill of the aneurismal varix. Before death, however, this tumor entirely disappeared, and nothing could be discovered in its situation, after death, but an enlarged, indurated, tuberculous liver. In Dr. Clark's case, a pulsating tumor existed in the epigastrium, with a thrill and a loud sawing sound. After death, the tumor was found to have been produced by the pancreas, of natural size, passing over the abdominal aorta, and brought into relief by the empty state of the stomach and intestines, and the thinness of the abdominal walls; the patient being in the last stage of tuberculous emaciation. These facts are not without a practical interest. You will remember the experiment I described to you some time ago, whenI was illustrating the mechanism of the blowing sound; when a ligature was passed round a gum-elastic tube so as to diminish its calibre at a certain point, and water was forced into the tube by a pump, that not only were vibrations and a blowing sound generated at the point of obstruction, but that they travelled most distinctly along the tube in the direction of the current, while they were hardly perceptible on the opposite side of the ligature. I mentioned also to you Dr. Corrigan's theory of the bellows sound being produced in unfilled arteries, and his mode of explaining the blowing sound in the tube beyond the ligature, the walls of which are rendered less tense by the obstruction, and his application of the same theory to the mechanism of the bellows murmur in aneurisms. The two cases of stricture of the aorta which I have related, and especially the apparent, but not real existence of an abdominal aneurism, seem to me singularly favorable to Dr. Corrigan's opinions.

The treatment of aneurism of the aorta is that of enlargement of the heart. In both diseases, the indications are plain and simple. You must resort to every possible means to restrain

the action of the heart, if it is at all disposed to be active. Passive exercise, or rest in many cases, a simple, unstimulating diet, a free condition of the bowels, freedom from mental anxiety and excitement, are plainly indicated. The use of sedatives, to relieve distress, is often necessary, and sometimes even the cautious use of blood-letting, especially by local means. The treatment of Valsalva, that by large and by repeated blood-letting, with very low diet, and perfect rest long continued, has been recommended by a high authority. But strong objections may be brought against its use. Frequent and free depletion, in addition to the danger of reaction, tends to render the blood thin, by depriving it of its fibrine. Thus the tendency to the formation of coagula in the aneurismal sac is lessened, and the chances of a cure are diminished; or if a cure is not to be expected, the chances of even checking the progress of the disease are less, since the formation of coagula, although they seldom effect a cure, are no doubt instrumental in delaying the fatal issue.

APPENDIX TO TUBERCLE AND CANCER.

As the heterologous deposits, tubercle and cancer, are fully discussed in the preceding lectures, and as the microscopic appearances that these deposits present are now attracting much attention, inasmuch as the microscopic pathologists regard a tubercle-cell and a cancer-cell as differing from all other cells, and therefore diagnostic of these interesting deposits, especially in cases in which the diagnosis by the more ordinary mode of examination is doubtful—I am induced to think that a translation from Lebert's work on Pathological Physiology, containing his description of the microscopic appearances of tubercle and of cancer, and illustrated by a selection from his excellent plates, will possess some value for the American student. I may add, that Lebert is at this time the highest authority in France on this subject, and is, perhaps, unsurpassed by any microscopist now in existence in microscopic pathology. The translation is a very free one, and I have condensed certain portions of the original, and omitted entirely other portions which did not seem necessary to a clear and brief account of these subjects.

The Microscopic Composition of Tubercle.

It is a general law in the molecular composition of morbid productions, that any thing which is really and materially different in pathology, exhibits this difference in the most minute elements of structure revealed by the microscope. The chief reason why this difference, which is so well marked in various morbid products, has not been admitted, is in part owing to the fact that medical microscopists have not examined the subject sufficiently in all its various relations, and partly because they have not employed a sufficiently high magnifying power in their investigations.

They have seldom employed a magnifying power of more than 300 diameters, but with this power, the small, primitive globules, which

often hardly attain to the size of one-hundredth part of a millimetre, resemble each other so much that their specific characteristics cannot be recognized. I have made all my observations with the large microscope of Oberhauser, a very perfect instrument, which allows a range from 25 diameters, to the highest range, 800 diameters, which is capable of being employed with precision. In this manner I have always been able to examine all the ordinary details of pathological anatomy with the greatest precision, and to carry the examination of molecules to the highest possible limit. I have measured the microscopic globules with a glass micrometer, which indicates the $\frac{4}{100}$ part of a millimetre, sometimes using another which indicates even the $\frac{8}{100}$ of a millimetre. Sometimes, also, I have employed the camera lucida to measure still more minutely, but this, however, is of no use.

The constant elements of tubercle are:

1st. A great number of molecular granules, perfectly round, of a grayish-white color, or with a slight yellow tint, sometimes compact, sometimes transparent in their centres, with a diameter of ·0012 to ·0025 of a millimetre. These granules completely surround the tubercle globule, so that it is often difficult to recognize it in the crude yellow tubercle. They are seen in much greater numbers, and quite disaggregated, in the softened tubercle.

2d. These granules, as also the tubercle globule, are united with each other by an intergranular, interglobular, hyaline substance, of considerable consistence, which serves as a cement to the elements of tubercle, and which becomes liquefied by softening.

3d. If the two elements which I have just described possess no peculiarities which belong to tubercle, and which do not distinguish it from other morbid products, there is a third element which is much more important, which, in fact, is entirely characteristic of, and peculiar to tubercle—*the tubercle globule, or cell.*

The form of the tubercle globules is seldom perfectly round, although it is probable that at the time of their excretion by the capillaries, they do assume a form more or less spherical, and that they afterwards assume a less regular shape, often becoming angular, on account of their close juxtaposition. Thus, as they commonly appear under the microscope (Pl. I., Figs. 1 and 2), especially in the crude tubercle, their outline is irregular, approaching sometimes to the sphere, sometimes to an oval, but generally they are irregularly angular and many-sided, with the angles and the edges rounded, as is very evident when they are suspended in water or in serum. Their color is a clear yellow, assuming a blackish

tint when a high magnifying power is employed. Their interior is irregular and of unequal consistence, which gives them a spotted appearance, independently of the granules which they may contain. But I have never been able to detect a true nucleus in these globules, although they sometimes present in their interior the appearance of an irregular vacuum, which resembles a nucleus. I have always examined this point with great attention, using the highest and the best defining magnifying powers, as well as different chemical reagents. We cannot consider the granules, which are irregularly distributed in the substance of the tubercle globule, as nuclei. These are only molecular granules, whose diameters seldom reach, and never exceed, ·0025 of a millimetre; often, indeed, they are not more than ·0012 to ·0015 of a millimetre. These granules, variable in number from 3, 5 to 10, or more, are not regularly distributed, and are not all visible in the same focus. The intergranular substance of the globules surrounds them, so that they are not ordinarily encompassed by a transparent areola. The interior of these granules appears opake.

The diameter of the tubercle globule varies. In the rounded globules, it ranges between ·005 and ·0075 of a millimetre, rarely extending to ·01 of a millimetre. The oval globules, as a mean, are ·0075 of a millimetre in length, and ·005 to ·006 of a millimetre in breadth. The diameter of the tubercle globule increases at the commencement of the period of softening.

The diameter of the tubercle globule varies within certain limits; but this variation is independent of age and of the tissue or organ in which the deposit has formed. It is more easily recognized in the yellow crude tubercle, than in the gray miliary granulation. In the recent tubercle, the tubercle globule is detected with difficulty, because it is concealed by the interglobular hyaline membrane which unites the globules, and by a large number of molecular granules which surround them.

It it important, therefore, in commencing the study of the tubercle globule, to select for examination a yellow cheesy tubercle, not too hard nor too soft, to disaggregate it with needles, in a drop of water, which can, however, never be done completely; and this difficulty is one of the most striking characteristics of the tuberculous deposit. It is well, after this has been done, to let the preparation dry a little between the plates of glass, in order that as many globules as possible may be seen at the same focus. The distinctness of the view may be increased by a fine diaphragm and by a good light. A lamp is, however, not as favorable for the examination of tubercle as the daylight; and if the lamp is em-

ployed, care must be taken that the light is not too strong. Having thus become familiar with all the details of the tubercle globule, it will be easily recognized whenever it is present. By this method, then, the tuberculous deposit can be readily distinguished from all other morbid products, a result which, in doubtful cases, no other mode of examination is capable of producing.

Water does not change the tubercle globule. Acetic acid renders it more transparent without changing it much, and establishes the absence of nuclei in its interior. (Pl. I., Fig. 3.) It is a very valuable mode of distinguishing the tubercle globule from other globules resembling it, except that they contain one or more nuclei. Acetic acid is especially useful in distinguishing the tubercle globule from the pus globule. Ether and alcohol react very slightly upon the tubercle globule. Strong ammonia renders it, at first, more transparent; it then dissolves the intergranular substance, and allows the molecular granules contained in it to become separated. A concentrated solution of caustic potassa completely dissolves the tubercle globule. The concentrated acids, especially the hydrochloric and the sulphuric acids, also dissolve it, but more slowly.

What is the position which the tubercle globule is entitled to occupy among the pathological cells? If it be true that a perfect cell is composed of an investing membrane, and of one or two nuclei and of nucleoli in the interior of these nuclei, yet I am convinced, from many observations of pathological cells, as well as of those found in healthy organs, that this mode of cell-formation is by no means universal, and only peculiar to a certain number of elementary globules. The tubercle globule appears to me to be one of the most simple forms of pathological cells, being composed of an enveloping membrane, containing a semi-liquid substance and a certain number of molecular granules irregularly scattered through it, as in the pyoid globule. This pyoid globule (Pl. I., Fig. 12, B), however (which is a variety of the development of the pus globule), differs from the tubercle globule in being more regularly spherical, more pale, more transparent, and by containing granules which are transparent in their centres, and seated in the periphery of the pyoid globule.

Let me pause here to consider a question important in a pathological point of view. Pathologists, of great merit and reputation, have regarded tuberculization as a modification of suppuration. On the other hand, they have often indicated the existence of tuberculous matter in the midst of cancerous tumors. With the naked eye, such mistakes could not be avoided. Thus, I have seen a substance which looked exactly like tubercle, but which contained nothing of the kind. I have seen a

mass which looked like cancer, but which disclosed, under the microscope, only the elements of tubercle. I have, however, in a certain number of cases, discovered the tuberculous matter and the cancerous matter in the same morbid product.

It is important, therefore, to possess certain positive and constant characteristics, by which the tuberculous deposit can be distinguished from the products of suppuration, and from the cancerous deposit. This desirable object, notwithstanding difficulties may sometimes exist, may be gained in a sure and positive manner, as I shall now explain.

Pus globules (Pl. I., Figs. 6 and 7) are larger than the tubercle globule. Their mean diameter is ·01 to ·0125 of a millimetre. They are not glued together as the tubercle globules are in their early stage; they are always found floating free in serum. Their shape is round and spherical, their surface is slightly rough, and is sometimes covered by molecular granules. Their investing membrane is more or less transparent. Their contents are more liquid, and you can notice in them, when they have attained to their full size, one, two, three, rarely four or five true nuclei, whose diameters are ·0033 to ·005 of a millimetre, and in the interior of which a nucleolus can often be detected.

With a high magnifying power it is easy to discover these nuclei without the aid of any chemical reagent. The acetic acid, however, renders them more distinct, while it discloses their absence in the tubercle globule. In certain kinds of pus, in which the globules are much changed by a serum of a bad character, this distinction is sometimes more difficult; but the shape, the free and disintegrated condition, and the size of the pus globule, will still serve to distinguish them. In another place I shall point out the characteristic differences between pus and softened tubercle, also the difference between tubercle and concrete pus.

The difference between the tubercle globule and the cancer globule is still more distinct. (Pl. I., Fig. 8.)

Not only the globules of cancer, but even their nuclei, are larger than the entire tubercle globule. The globules of scirrhus have a diameter of ·0175 to ·02 of a millimetre, and sometimes of ·025 of a millimetre. Their outline is regular, their appearance pale, and their surface is finely dotted with minute granules, which are situated between the investing membrane and the nucleus. This nucleus is commonly single, but sometimes double, and with a strongly marked outline, round or oval, and with a diameter of from ·0125 to ·015 of a millimetre. These nuclei are often found freed from their investing membrane. When this is the case, and

a large number of these free nuclei are clustered together, they resemble somewhat tubercle globules, but the differences in the diameters, in the outline, in the central substance, and in the existence of a certain number of perfect cancerous globules, will remove any doubt that may exist.

The globules of encephaloid, which are very like those of scirrhus; or rather the nucleus of the true encephaloid globule—for authors have generally mistaken the nucleus for the perfect globule—has a diameter from ·01 to ·015 of a millimetre. Its shape is a very regular sphere, or oval, with a marked outline finely shaded all around its internal circumference, containing besides a fine granular matter, one, two, rarely three round nucleoli, with diameters of from ·0025 to ·0033 of a millimetre, and transparent at the centre. A fact which establishes the diagnosis still more clearly is, that when the globules are perfectly formed, they are surrounded by an investing membrane, which is often irregular in shape. The whole globule thus represented has a diameter of ·015 to ·02 of a millimetre, and sometimes even of ·035 of a millimetre, and possesses characteristics peculiar to itself.

The crude tubercle, then, contains an element which is peculiar to it, and which distinguishes it from all other morbid products.

I will now pass to the study of the softened tubercle, limiting myself, for the present, to indicating the physical changes in the softened tubercle as revealed by the microscope, and reserving the physiological explanation for another place. In order to appreciate properly the changes which take place during the softening of the tuberculous matter, the use of the microscope is indispensable, for the reason, that the parts surrounding the tubercles often inflame and secrete pus, and then the elements of suppuration are mixed with those of tubercle. As the naked eye cannot discover all these details, much confusion would exist without the aid of the microscope.

I may say, in general terms, that the principal change that occurs in the tubercle while softening, consists in the liquefaction of the interglobular hyaline substance, which is sufficiently solid and consistent in the crude tubercle to hold the tubercle globules in close union. But in the softened tubercle, they become disaggregated, separated, although clustered groups may still be discovered. As the globules become free, they become more rounded, almost spherical; they become, at the same time, more transparent and more thin, and the blastema which surrounds them becomes more granular. (Pl. I., Figs. 4 and 5.)

Both by the naked eye and by the microscope, pus is frequently found

united with softened tubercle. (Pl. I., Fig. 9, and Fig. 12, B.) It would appear that the presence of pus hastens the decomposition of the tubercle globule, and this is one reason why the matter contained in tuberculous ulcers is so often without tubercle globules.

Finally, it may be stated, that the tubercle globule disappears in a nearly perfect dissolution, after having been disaggregated into granules. These globules, then, undergo three phases of development. They are at first closely packed together, and compact in their interior. Then they separate from each other and increase in size, which, instead of being owing to a more perfect development, is, in fact, the commencement of decompostion, and is owing to an endosmosis of the surrounding blastema, which becomes more and more liquid. At last, these little globules, whose internal and molecular cohesion has already been disturbed, finally, by running together, form a yellow and a more or less liquid mass.

There is some analogy in the mode in which the pus globule and the tubercle globule disappear. The former is disintegrated into granules before it can be absorbed.

If the crude tubercle and the softened tubercle constitute the two first stages of this deposit, and the diffluence of the elements of tubercle the third stage of its evolution, there is still a fourth stage,—its passage into a cretaceous state. I can confirm the opinion that this cretaceous transformation of tubercle is one of the most powerful means which nature employs to cure the tuberculous disease. Its microscopic composition is altogether in favor of this view of the question. At the commencement of this change, we can still recognize a considerable number of tubercle globules, and with them a kind of mineral dust formed of very fine granules, whose diameters are from ·001 to ·0015 of a millimetre, transparent in the centre, looking black under a high magnifying power, but under a low power, as well as by the naked eye, having a yellowish-white tint, and being more resistant to compression than the soft elements of ordinary tubercle. These latter elements diminish in proportion as the granular, amorphous, mineral elements increase. They become more solid and dry, as the portions capable of dissolution are absorbed. The cretaceous tubercle often contains much black pigment, and many times I have met with a considerable number of crystals of cholesterine. (Pl. I., Fig. 10.)

Having described the elements which are essential to tubercle, I will next examine other elements which are not essential, but yet are of frequent occurrence.

The pigment infiltration, or melanosis, which is also met with in many other morbid products, appears in three different forms. 1st. As a gran-

ular infiltration. 2d. As the contents of certain globules, having a diameter from ·016 to ·024 of a millimetre, and sometimes reaching ·033 of a millimetre. (Pl. I., Fig. 11.) 3d. As fine granules contained in certain normal, or pathological cells. Thus it is frequently contained in epithelial cells, and expectorated in abundance.

This pigment is also found surrounding pulmonary tubercles, as the gray granulation, the cretaceous tubercle, and tuberculous excavations. It is also often found in abundance in the bronchial glands. It is sometimes noticed in the mucous membrane of the intestine, and especially around tubercles of the peritoneum. It is a carbonaceous substance.

Fat, in the form of fat vesicles, is frequently found in tubercles. (Pl. I., Fig. 13, C.)

It is not uncommon to find fibres in tubercle, but they very rarely belong to the tuberculous secretion. Generally, they are fibres of the tissue of the organ in which tubercle is secreted. Thus, the gray, semi-transparent tubercle in the lungs, often contains the elastic fibres of the cellular tissue of the lungs.

In certain rare cases, crystals exist in tubercle. Once I met with three-sided prisms in tuberculous matter from the lungs; another time, in the bronchial glands; and in a third case, rhomboidal plates of cholesterine in softened tubercle in the neck, and which was not cretaceous.

Once I have met with large globules of a greenish-brown color, ·016 to ·025 of a millimetre in diameter, and containing three or four small globules. I have found, also, mixed with the elements of tubercle, certain accidental products derived from the surrounding tissues. I have already mentioned the pus globule. Other globules frequently met with, are the product of the surrounding pulmonary inflammation. These are globules with a diameter of ·02 to ·025 of a millimetre, filled with yellowish granules, and sometimes containing a nucleus. (Pl. I., Fig. 12, C.)

Another element not unfrequently met with, and which might easily lead to mistakes, are young epithelial cells (Pl. I., Fig. 12, A.), derived from the capillary bronchi when the lung is cut, having a diameter of ·0125 to ·015 of a millimetre. These are of an irregularly rounded shape, containing a nucleus with a diameter of ·005 of a millimetre, and which sometimes contains a nucleolus, or a finely granulated matter. These cells are found in considerable number around agglomerated masses of tubercle globules, but never in the midst of them, so long as they are united by the intercellular hyaline substance. By the side of these round or oval young epithelial cells, are found the cylindric epithelial scales, with

or without vibratile cilia, and which could not easily be mistaken for tubercle globules.

In conclusion, we find as the constant and essential elements of tubercle, granules, and an interglobular hyaline substance, and globules peculiar to tubercle. After its excretion, the tubercle first assumes a compact form, then it softens, and at a still later period it dissolves; or it withers and becomes cretaceous. The elements which are not constant, but which are found more or less frequently in tubercle, are melanosis or black pigment, which is the most common, fat, fibres, globules of a decided color, and finally crystals, commonly those of cholesterine.

As elements accidentally mixed with tubercles, we often find under the microscope different products of inflammation, of exudation, of suppuration, and of the epithelial secretion, globules of different kinds, which come from the tissues surrounding the tubercle, but which are never met with in the midst of its elements.

In the gray semi-transparent granulation of the lungs, we always find a mixture of areolar fibres with a grayish hyaline substance and with tubercle globules. The fibres are composed of the elastic fibres of the pulmonary cellular tissue. The gray tint of the granulation is sometimes heightened by the admixture of the black pigment.

The yellow opake tubercle is identically the same as the gray semi-transparent tubercle, only in the latter, the tubercle globules are smaller, and more closely packed in the substance which surrounds them. The yellowish aspect is produced by the confluence and increased size and abundance of the tubercle globules after the destruction of the surrounding fibres which tended to separate them, and at the same time the hyaline membrane becomes more opake and granular.

The gray, semi-transparent granulation is not the constant or the necessary commencement of the tuberculous deposit. It may occur originally as the yellow opake granulation. Very small yellowish points make their appearance, in which the microscope discovers a few fibres, much less numerous than in the gray granulation. Their principal element is the tubercle globule, and the interglobular hyaline membrane is granular, and with very little transparency.

The liquid which covers the internal aspect of tuberculous excavations contains, sometimes, tubercle globules in their perfect form, but generally they are more or less distended by the softening that has taken place, and most of them are in a state of diffluence. It also contains pus globules, the large granular globules of inflammation, a viscid mucous fluid,

blood globules, pulmonary fibres, black pigment, epithelial scales, three-sided prisms, and fat vesicles.

Under this liquid layer, composed of so many elements, are false membranes, composed of a fibrous, stratified substance, and containing numerous pus globules.

Beneath this layer of fibrine is the true lining membrane of the excavation—it is organized and vascular. Its structure is irregularly fibrous, and among the fibres are numerous small globules. Sometimes it contains but very few blood-vessels, and then the fibrous tissue is dense, white, and very abundant, appearing like cartilage. But I have never found in it the slightest traces of the elements of cartilage.

The microscopic examination of the expectoration in tuberculous phthisis discloses the following facts: The matter contains, in the first place, substances which are not at all specific, as saliva mixed with mucus and epithelial scales from the mouth, which latter are sometimes quite abundant; epithelial scales from the bronchi, mucus, vibriones, blood globules, crystals, black pigment, globules of fat, granular globules, and pus globules.

Besides these are noticed small masses or little pellicles, which at first sight might be mistaken for tuberculous matter. The microscope, however, only discloses globules of pus and a granular coagulation. These are probably false membranes coming from tuberculous cavities. Again, we notice masses like the preceding in appearance, in which the microscope only discloses numerous molecular granules, which are probably produced by diffluent tuberculous matter: again, there is noticed amorphous mineral granules, which, perhaps, come from cretaceous tubercles. And finally, we may meet with the true tubercle globule. But this is very rare. I am not sure that I have ever met with it so distinctly that its existence was not doubtful. Sometimes pulmonary fibres are found in the expectoration. There is, then, nothing specific in the tuberculous expectoration.

The Microscopic Elements of Cancer.

Authors of much merit have denied that the microscopic elements of cancer were characteristic. I have arrived at an opposite conclusion, and I maintain that the cancer globule has striking characteristics which distinguish it from every other form of morbid product. It must not be forgotten, that there are certain general forms of cells and of nuclei, the types of which are met with in very different products. But this I maintain, that the different pathological products which are composed of

elementary globules, individually present certain characteristics by which they can be distinguished by those somewhat accustomed to the use of the microscope. I will go even further, and state that the cancer globule is one of the cells which possess the most striking characteristic features to distinguish it from every other kind of cell. It is important, however, to add, that the cancer globule is subject to very many variations; but I hope by pointing out these varieties carefully, and at the same time by explaining the sources of mistake, and the difficulties in the diagnosis, to place before the reader their peculiar characteristics.

The cancer globule is composed of an enveloping membrane, and a nucleus which contains nucleoli. (Pl. II., Fig. 1.) The diameter of the external cell varies in different cases. Its mean diameter is ·02 of a millimetre, sometimes it is only ·015 of a millimetre. Very often it is much greater, extending to ·03 of a millimetre, or even beyond that point. Its shape is round or ovoid—round more frequently in the globule of encephaloid, a little elongated in the globule of scirrhus. In many cases it is easy to trace the progress of one of these forms as it passes into the other form. Very frequently this external enveloping membrane assumes many different forms. It is generally more flattened than the nucleus. Sometimes it is pale, and perfectly transparent. At other times it is covered by fine dots, and quite frequently it is so filled with granules that it exactly resembles the large granular globules of inflammation. (Pl. II., Fig. 2.) It is also not uncommon to meet with both regular and irregular globules which contain a certain number of nuclei, and we may discover large parent cells, with a diameter reaching even to ·05 of a millimetre, of a rounded or oval shape, and which contain four, five, six, or even a greater number of nuclei. (Pl. II., Fig. 3.) At other times we meet with large membranous expansions, in which we can distinguish a considerable number of nuclei, surrounded by a granular and dotted mass. (Pl. II., Fig. 4.)

The nuclei vary in their diameters from ·0075 to ·02 of a millimetre. The smaller are found chiefly in the perfect globules of scirrhus; the large round, or elliptical nuclei, with diameters extending from ·015 to ·02 of a millimetre, are principally found in the encephaloid cancer. In some forms of cancer these nuclei constitute so decidedly the predominating element under the microscope, that we might be tempted to assume that they were the type of the cancer globule, did we not observe these same globules in their more perfect form, that is, with their enveloping membrane, in other cases of cancer. (Pl. II., Fig. 5.) These nuclei are sometimes very pale: at other times, and this is especially the

case in scirrhus, their outline is very distinct. In many cases of encephaloid they present a characteristic shading at their whole circumference. In a certain number of cases the enveloping membrane of the cancer globule is elongated, pointed at each end, and even at several points of its circumference. It then bears some resemblance to the fusiform fibro-plastic bodies. It can always, however, be readily distinguished from these bodies by its much greater size, by being much less elongated, and by its characteristic nuclei and nucleoli. (Pl. II., Fig. 6.)

If the nuclei and nucleoli of the cancer globule always possessed the distinct form which I have just described, nothing could be more easy than to detect them by a microscopic examination. But, as it generally happens that cancer is mixed with much fatty matter, the nuclei are found to undergo different changes on this account. Thus we often find them filled with granules and small grumous masses; sometimes, indeed, they are infiltrated with a homogeneous and confluent fatty matter. (Pl. II., Fig. 7.)

The nucleoli have a diameter which varies from ·0025 to ·0033 of a millimetre, and even to ·01 of a millimetre. Their number is from one to five. But as the nuclei which contain them are somewhat thick and spherical, we cannot recognize them all under the microscope at the same focal distance. These nucleoli have a peculiar character. Their outline is distinct, but their interior is seldom transparent—ordinarily it is dull and homogeneous. I was for a long time in doubt what these nucleoli were; but I have recently discovered that they are imperfectly developed nuclei. In examining some large nucleoli under a magnifying power of 1000 diameters, I saw that they contained two or three secondary nucleoli. (Pl. II., Fig. 8.)

It is not uncommon to meet, in cancer, with large concentric cells with a diameter from ·04 to ·05 of a millimetre, and with thick walls inclosing many concentric globules. (Pl. II., Fig. 9.)

The cancer globule appears to me to be formed in this way: The capillaries excrete the cancerous matter in a liquid state. In this liquid, nuclei form, and soon after nucleoli. Possibly the nucleoli may form first. Around the nucleus, molecules of the liquid blastema first excreted arrange themselves, so as to form irregular enveloping shreds, or regular rounded or oval globules. It may possibly be the case that these concentric globules are only ordinary cancer globules, all the portions of which are remarkably developed. I have also seen the cancer globules assume the appearance of clustering when they were filled with granules

of fat, and when the nuclei also were deformed by the infiltration of fatty matter.

It is not reasonable to suppose that the cancer globules, which are first secreted, continue to exist for a long time. After a time they become deformed, they lose their distinct outline, and are finally dissolved into granules. At the same time, the excreted blastema which is constantly being poured out by the vessels, forms new cells. Thus, a certain number of the cancer globules appear incompletely developed, others are well developed, and a certain number is undergoing decomposition.

The cancer globule of scirrhus is ordinarily furnished with an enveloping membrane, which is round, ovoid, or irregular in shape. Its mean diameter varies from ·015 to ·02 of a millimetre. It is finely dotted all around the nucleus. This nucleus is small, its diameter varying from ·0075 to ·01 of a millimetre. Its outline is very sharp, and it exhibits, in its interior, granules and little masses (grumeaux), and sometimes nucleoli. (Pl. II., Fig. 1, *b b*.)

The cancer globule of encephaloid is surrounded by an envelope, regular or irregular in shape, having a diameter between ·02 and ·03 of a millimetre. The nucleus is spherical, or very often elliptical, pale, shaded at its circumference, and containing from one to three very distinct nucleoli. Generally, as already stated, the nuclei are seen under the microscope in greater number than the perfect cells. (Pl. II., Fig. 1, *a a*.) Frequently every form intermediate to these two types of the cancer globule will be noticed.

Next to the cancer globule, which is the characteristic element, is the fibrous element, which is sometimes the predominating element. It presents very different appearances in different cases. In scirrhus, it is formed by a network of fibres arranged in bundles, which cross each other in every direction, and communicate with each other by fibres, which pass from one bundle to another. (Pl. II., Fig. 10.) The primitive fibres, in this case, are well defined. They are delicate, and do not exceed in breadth the ·0025 of a millimetre. They are generally less tortuous than the fibres of ordinary cellular tissue. In some cases these fibres interlace with each other without being arranged in bundles. (Pl. II., Fig. 11.) In certain organs, especially in cancer of the mamma, there are numerous elastic fibres. In some exceptional cases, I have met with a fibroid network, inclosing in its meshes cancer globules and resembling exactly coagulated fibrine. In the soft, encephaloid cancer, the fibres are pale and delicate, and much less numerous than in scirrhus. (Pl. II., Fig. 12.) Nevertheless, I have met with cases of med-

ullary cancer, in which the encephaloid matter was inclosed in a dense and fasciculated fibrous stroma.

Fusiform bodies, such as are met with in other morbid products, are very frequently seen in cancer. They are distinguished from the fusiform cancer globule by the difference in their nuclei, that of the cancer globules being much larger. (Pl. I., Fig. 12, D.)

These fibres, these fusiform fibro-plastic globules are formed from the exuded blastema, as is also fat, pigment, and other substances.

After the cancer globules, the fibres, and the fusiform bodies, the substance which is met with most frequently and abundantly in cancer, is fat. It is seldom absent, and it is sometimes so abundant and so mixed with the cancer globules, that they can hardly be distinguished. The fatty element occurs under the forms of granules, of free fat vesicles (Pl. I., Fig. 13), fatty spots, and cholesterine. The granules (Pl. II., Fig. 5, *d d*) are commonly found in abundance outside the cancer globules, but very often also they exist in their interior, and then we can distinctly trace the change from a simple cancer globule to that which resembles exactly the large granular globules of inflammation. Frequently, these granules are deposited in the nuclei of encephaloid globules. But that which renders these globules not easily recognizable, is the fact that fat is frequently deposited in them which is confluent and homogeneous in its character. Their outline is thus altered, and it requires great attention to distinguish them. It is these globules which constitute the fatty matter which looks like tubercle, an appearance noticed especially in sarcocele.

Large granular globules (Pl. I., Fig. 12, C) analogous to those noticed as the product of inflammation, with a diameter of from ·02 to ·03 of a millimetre, are commonly noticed in cancer. I have already stated that the cancer globule when infiltrated with fat, may assume the appearance of these inflammatory globules. But I think that the true inflammatory globule is also often found in cancer. When it is examined by a low power and by direct light, it appears in groups of a dull white or yellowish aspect. With a high power, and by reflected light, it appears of a blackish-brown color. It is usually so spherical, that it can be burst by compression, and made to discharge numerous granules. These globules are found in all kinds of cancer. I have sometimes seen them existing as a general infiltration into the cancerous mass, and sometimes forming a network of a dull-white color, constituting the reticulated figures so well described by Muller. They can sometimes be enucleated and studied separately.

The black pigment or melanosis, both in the form of granules and of globules, is found in both scirrhus and encephaloid cancers. (Pl. I., Fig. 11, *a b*.)

I have also noticed a peculiar coloring matter, of a yellowish tint, which I have named *Xanthosis*. It varies from a saffron to an orange tint. It appears to be a kind of fatty matter.

Crystals of cholesterine is one of the most common elements of cancer. I have also seen prismatic needles in cancerous deposits; also, mineral concretions, amorphous, or bone-like, yet without the structure of bone.

All the forms of cancer present the evidences of vascularity.

The colloid or jelly-like cancer is as well recognized a form of the disease as scirrhus or encephaloid. The cancerous tissue, especially the encephaloid, sometimes constitutes the base of the tumor, and then the cancer globules are only found in the deeper portions. In this case the gelatinous matter does not contain the true elements of cancer. We find a network of fibres forming large areolæ, and filled by a transparent matter, containing pale, granular globules. This colloid matter does not appear to be different from that noticed as the product of inflammation, or as the contents of various kinds of benignant tumors. It only differs from it by being combined with encephaloid. But, in other cases, these areolæ are filled by large cells or semi-transparent lobules, which contain numerous cancer globules and nuclei.

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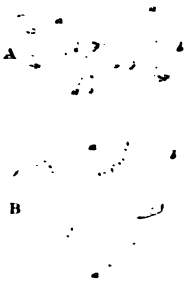

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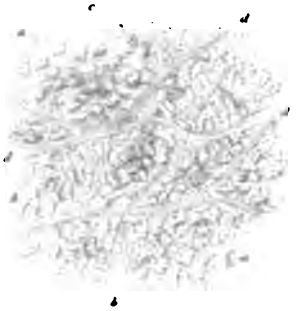

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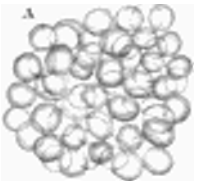

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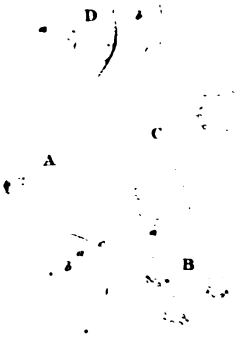

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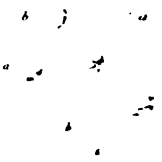

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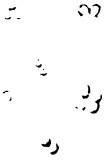

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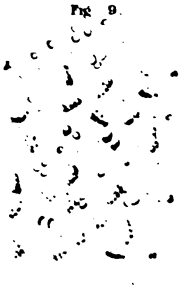

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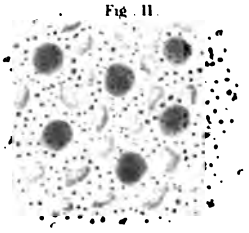


Fig. 4.

EXPLANATION OF PLATES.

PLATE I.—TUBERCLE.

FIG. 1. A.—Tubercle globules as seen with a magnifying power of 400 diameters.

FIG. 1. B.—The same globules as seen with a magnifying power of 600 diameters. In A and B the tubercle globules are designated by the letter *a*; the letter *b* indicates the granules which surround these globules.

FIG. 2.—Tuberculous matter in the midst of the pulmonary fibres.
a a, Tubercle globules; *b b*, molecular granules; *c c*, interglobular hyaline substance; *d d*, pulmonary fibres.

FIG. 3.—Tubercle globules rendered transparent by acetic acid.

FIGS. 4 and 5.—Globules of softened tubercle. The tubercle globules and the granules are disaggregated, with a tendency to assume a rounded form.
a a, Tubercle globules; *b b*, granules.

FIGS. 6 and 7.—Pus globules; intended to show the difference between them and tubercle globules.

FIG. 6.—Globules of pus in their ordinary condition.
a a, Pus globules without much transparency; *b b*, pus globules which display their nuclei.

FIG. 7.—Pus globules treated with acetic acid.

FIG. 8.—Cancer globules, intended to show the difference between them and tubercle globules.
a, Walls of the cancer-cell; *b*, the nucleus; *c*, the nucleolus.

FIG. 9.—Tuberculous matter infiltrated with pus and treated with acetic acid.
a a, Tubercle globules, these contain no nuclei; *b b*, pus globules, these contain several nuclei; *c c*, nuclei of pus globules, their enveloping cell having been dissolved by the acetic acid.

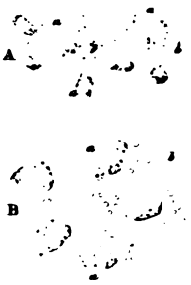
FIG. 10.—Cretaceous tubercle.
a a, Tubercle globules; *b b*, crystals of cholesterine; *c c*, granules of tubercle.

FIG. 11.—Tuberculous matter mixed with melanosis.
a a, Melanotic globules; *b b*, melanotic granules; *c c*, tubercle globules.

FIG. 12.—Exhibits several forms of globule which may be mistaken for the tubercle globule or for the cancer globule.
A, Young epithelial cells, which may be mistaken for tubercle globules.
a, Wall of the cell; *b*, nucleus; *c*, nucleolus.
B, Pyoid globules, an elementary form of pus globule, which contain from four to ten or more granules, but no nuclei. They are larger and more spherical than the tubercle globule.
C, Granular globules of inflammatory exudation, with (*a*) and without nuclei. They are distinguished by their large size.
D, Fibro-plastic globules of inflammatory exudation, which might be mistaken for the form of the cancer globule exhibited in Pl. II., Fig. 6, but which are distinguished from them by their nuclei.
a, Fibro-plastic globule; *b*, the same globule more advanced and elongated.

FIG. 13. A.—Fat vesicles, constituting the fatty tissue.
B, Fat vesicles contained in the secreting cells of the liver.
a, Walls of the liver-cells; *b*, fat vesicles; *c*, nuclei of the liver-cells.
C, Isolated fat globules.

Fig 1



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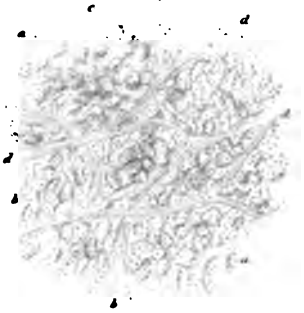

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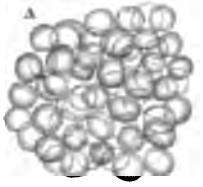

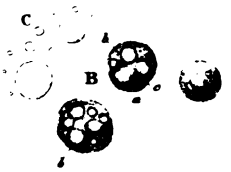

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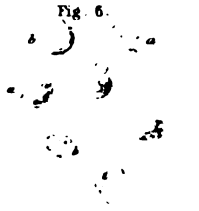

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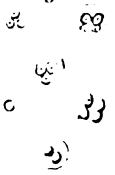

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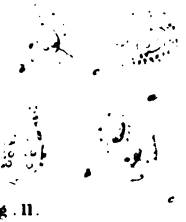

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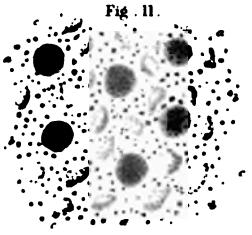

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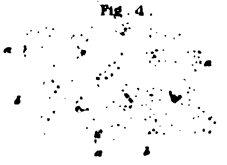

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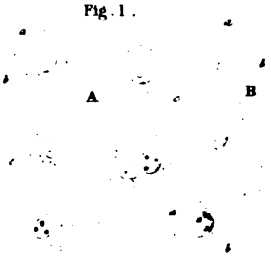

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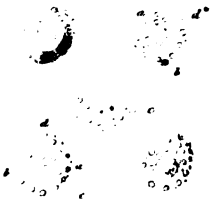

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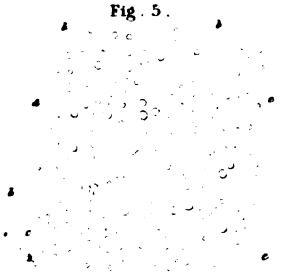

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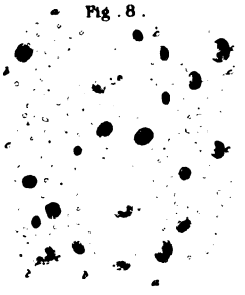

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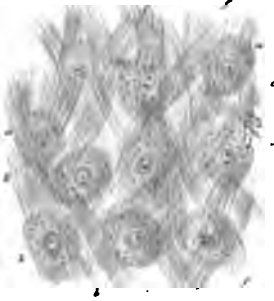

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Fig. 12.

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PLATE II.—CANCER.

FIG. 1.—Cancer globules.
A, Cancer globules with a large nucleus containing nucleoli.
a a, Walls of the cancer globule; *b b*, nuclei; *c c*, nucleoli.
B, Very large cancer globules, containing a very small nucleus.
a a, Walls of the cancer globules; *b b*, nuclei.

FIG. 2.—Cancer globules becoming granular.
a a, Walls of the cancer globule; *b b*, nuclei; *c c*, nucleoli; *d d*, granules.

FIG. 3.—Parent cancer globules, containing many nuclei.
a a, Walls of the cancer globule; *b b*, nuclei; *c c*, nucleoli.

FIG. 4.—A membranous expansion containing numerous nuclei of the cancer globule.
b b, Nuclei; *c c*, nucleoli; *d d*, granules; *e e*, intercellular substance.

FIG. 5.—Cancerous juice containing the nuclei of the cancer globule and granules, but no perfect cancer globules.
b b, Nuclei; *c c*, nucleoli; *d d*, granules, and small fat vesicles.

FIG. 6.—Fusiform cancer globules.
a a, Walls of the cancer globule; *b b*, nuclei; *c c*, nucleoli.

FIG. 7.—Nuclei of cancer globules infiltrated with fat, under different forms.
b b, Nuclei; *c c*, nucleoli; *d d*, irregular granules.

FIG. 8.—Cancer globules exhibiting nucleoli and secondary nucleoli.
a a, Walls of the cancer globule; *b b*, nuclei; *c c*, nucleoli; *c' c'*, secondary nucleoli.

FIG. 9.—Concentric cancer globules.
a a, External walls of the cancer globule; *a' a'*, internal wall of the cancer globule; *b b*, nuclei; *c c*, nucleoli.

FIG. 10.—Cancerous tissue, exhibiting numerous fibres in bundles.
a a, Walls of the cancer globules; *b b*, nuclei; *c c*, nucleoli; *f f*, bundles of fibres.

FIG. 11.—Cancerous tissue exhibiting fibres which are not arranged in bundles.
a a, Walls of the cancer globules; *b b*, nuclei; *c c*, nucleoli; *f f*, fibres.

FIG. 12.—Cancerous tissue exhibiting fibres which are pale and few in number.
a a, Walls of the cancer-cell; *b b*, nuclei; *c c*, nucleoli; *f f*, fibres.

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